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Volume 238 FEBRUARY 19, 1948 Number 8

WHICH TYPE CESAREAN SECTION?*

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BROOKLINE, MASSACHUSETTS

ABDOMINAL delivery may be accomplished in one of three ways: by the classic technic, by the low-segment operation or by some form of extraperitoneal cesarean section. It is not possible to compare absolutely the safety of the various procedures because of the variance in skill of individual operators and hospital equipment and also because the results reported by individual operators and clinics are not standardized so far as the condition of the patient at the time a particular technic was chosen for abdominal delivery is concerned.

The classic method results in the highest morbidity and mortality because the uterine incision is the only barrier between the peritoneal cavity and the interior of the uterus, which, as repeated studies have shown, may be invaded by pathogenic bacteria soon after delivery. The classic technic tends to form more adhesions than the other types, and rupture of the uterus occurs more frequently in subsequent pregnancies. The low-cervical operation has a better record. With the development of a bladder flap there is a double barrier to the spread of infected uterine contents. The uterine incision is well covered by peritoneum, adhesions are less likely to form, and the incidence of future rupture is less. We have collected a series of 14,776 low-segment operations reported in the literature in which the mortality was 1.52 per cent, whereas 15,030 patients subjected to classic sections by the same operators and under similar hospital conditions had a mortality of 3.87 per cent (Table 1). It is therefore apparent that, in general, the lower-segment operation is over twice as safe as the classic and has fewer subsequent complications. These figures are even more impressive when it is considered that the low-segment operation was frequently selected because the patient was potentially infected. The low-segment or low-cervical cesarean section is usually a safe and satisfactory procedure when done electively, when patients have been in labor for ten hours or less, with intact membranes, and when there have been no vaginal examinations. Since the incision in the uterus can be thoroughly covered by peritoneum there is very little danger of peritonitis. But, when the membranes have been ruptured or labor has been in progress for several hours or when there have been any vaginal or many rectal examinations, any form of intraperitoneal cesarean section may be fraught with danger, and

TABLE 1. *Mortality in Low-Cervical Compared with That in Classic Section.*

AUTHOR	LOW-CERVICAL SECTION		CLASSIC SECTION	
	NO. OF CASES	MORTALITY %	NO. OF CASES	MORTALITY %
Skeel and Jordan[1]	108	2.8	827	7.6
Phaneuf[2]	166	0.6		
Montgomery[3]	33	3.1	180	5.5
Sackett[4]	224	2.7	303	4.9
Greenhill[5]	108	0.0		
Lull[6]	103	3.8	478	6.7
Waters and Leavitt[7]	116	4.2	533	5.7
Seely[8]	87	0.0	105	7.6
Daily[9]	461	0.6	8	25.0
Maxwell[10]	165	0.0	113	6.1
O'Connor[11]	133	5.3	296	4.0
Courtiss and Fischer[12]	376	1.3	623	7.7
Arnot[13]	204	0.0	151	4.6
Garrison[14]			107	1.9
Bickel[15]	183	2.7	116	14.6
Gernez et al.[16]	131	0.0		
Thompson et al.[17]	955	1.2	618	3.6
Stude[18]	74	4.0	70	5.7
Barrett[19]	620	0.8	235	3.4
DeNormandie[20]	1112	2.6	870	3.2
DeLee[21]	541	2.0	490	5.5
DeLee[21]	1875	0.9	168	6.0
Ryder[22]	100	2.0	218	0.9
Lazard[23]	243	0.4	54	9.3
Gustafson[24]	208	0.9		
Irving[25]	686	0.9	1031	1.3
Falls[26]	57	0.0	57	1.7
Gustafson[27]	135	3.7	226	8.4
Mathews et al.[28]	264	1.8	737	3.9
Manahan et al.[29]	218	1.8	894	1.7
Rosensohn et al.[30]	215	2.3	255	3.9
King[31]	488	3.7	545	7.1
Lull[32]	324	1.2	550	3.2
Hawks[33]	30	0.0	492	4.2
DeNormandie[34]	1336	1.2	1009	2.9
DeNormandie[35]	1206	2.1	934	2.5
DeNormandie[36]	1003	2.3	898	2.9
Quigley[37]	196	1.0	717	3.3
Falls[38]	142	0.0	122	1.6
Heffernan[39]	150	0.0		
Totals	14,776		15,030	
Percentage mortality		1.52		3.87

*Presented at the annual meeting of the Massachusetts Medical Society, Boston, May 21, 1947.

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in such cases the lower-segment operation does not offer sufficient protection against peritonitis.

Seventy-five per cent of deaths from cesarean section occur from infection or hemorrhage, and the remainder from the complications of major surgery or conditions existing prior to the section and possibly aggravated by it. Peritonitis can occur without hemorrhage, which, when present, always predisposes to infection. The advances of chemotherapy and the development of the antibiotics will control most infections, but mothers still die from peritonitis in spite of adequate treatment by these agents. Many of these patients have been attacked by organisms, such as the anaerobic nonhemolytic streptococcus, that are resistant to these drugs. In a series of 1887 cesarean sections reported by Irving[25] from the Boston Lying-in Hospital, covering a period of ten years from 1934 to 1943, 42 per cent of the total number of deaths were due to peritonitis.

When actual signs of infection, such as fever, a rapid pulse and a foul amniotic discharge, are present before delivery, any type of transperitoneal section is definitely contraindicated. In 1933 DeLee[40] wrote: "My opinion is firm . . . laparotrachelotomy for all, but in those where infection is suspected, then Latzkos, Porro, or craniotomy."

To consider these procedures in reverse order, craniotomy on the living child is mentioned only to be vehemently condemned. It has no place in modern obstetrics, and the claim that sacrifice of the baby saves the mother is disproved by the experience of Baird,[41] who reports a maternal mortality of 7 per cent in 147 cases of craniotomy. Cesarean hysterectomy for infection adds operative shock and additional blood loss to that of a cesarean section, and if the patient is a young primipara the loss of the uterus may indeed be tragic. We have collected from the literature a series of 119 cesarean hysterectomies done for infection, with a mortality of 14.2 per cent. (Table 2). We believe that cesarean hysterectomy has no place in the treatment of infected patients and should be reserved for parturient patients with fibroids, atonic uteri or other pathologic conditions of the uterus.

Although the morbidity and mortality for abdominal delivery in cases of infection, both potential and actual, have improved since the advent of the antibiotics, chemotherapy and well stocked blood banks, the relative safety of the classic, low-segment and extraperitoneal types of section remains the same. When potential or actual infection exists neither the classic nor the lower-segment operation, even with the antibiotics and chemotherapy, affords the patient the full measure of protection.

The extraperitoneal approach, the principles of which were first expounded over a hundred years ago, is the safest method of abdominal delivery. In 1821 Ritgen[51] first tried to approach the lower anterior extraperitoneal surface of the uterus from the lateral aspect of the bladder through the paravesical space. Fundamentally the operations of Baudeloque,[52] Thomas[53] and Latzko[54, 55] fall into the same group. The commonly accepted technic, until the presentation of Waters[56] and Ricci,[57] was

TABLE 2. *Cesarean Hysterectomy for Infection.*

AUTHOR	NO. OF CASES	NO. OF DEATHS
Greenhill[42]	1	0
Hawks[43]	2	1
Lash and Cummings[45]	12	4
Adair[44]	8	0
Phaneuf[46]	7	0
Lazard[46]	1	0
Lazard[47]	9	2
Daily[9]	30	0
Maxwell[10]	2	1
Arnot[12]	10	2
Briscoe[48]	3	3
Wilson[49]	9	0
Stude[50]	2	1
Irving[25]	21	3
Falls[26]	2	0
Totals	119	17 (14.2%)

TABLE 3. *Extraperitoneal Latzko Cesarean Section.*

AUTHOR	NO. OF CASES	NO. OF DEATHS
Cosgrove[58]	74	2
Steele[59]	59	5
Aldridge[60]	27	1
Hawks[43]	30	0
Irwin[61]	285	9
Burns[62]	79	2
Sackett[4]	51	4
Perrins[63]	22	0
Holtermann[64]	194	10
Norton[65]	26	0
Fleischer and Kushner[66]	19	0
DeNormandie[28]	13	0
DeNormandie[28]	33	2
Irving[25]	40	1
Waters[67]	193	3
Totals	1145	39 (3.4%)

TABLE 4. *Modern Extraperitoneal Cesarean Section.*

AUTHOR	NO. OF CASES	NO. OF DEATHS	DEATHS FROM SEPSIS	DEATHS FROM HEMORRHAGE
Norton[73]	160	3	2	1
Ricci and Marr[73]	175	1	0	1
Irwin[61]	32	0	0	0
Pieri and Irving[74]	20	0	0	0
Eisaman and Austin[75]	22	0	0	0
Williamson and Goldblatt[76]	25	0	0	0
Daichman and Pomerance[77]	100	0	0	0
Bowles[78]	10	0	0	0
Burgeois[79]	10	0	0	0
Briscoe[48]	9	0	0	0
Stearns[80]	16	0	0	0
Crass[81]	89	1	1	0
Irving[25]	59	2	–	–
Waters[67]	290	2	1	–
Heffernan and Sullivan	72	0	0	0
Totals	1089	9 (0.82%)	4	2

that described by Latzko. We have collected from the literature a series of 1145 cases in which Latzko sections were performed, with a mortality of 3.4 per cent (Table 3). Although, as noted by the authors, the collections of classic and low-segment operations contained an admixture of clean, potentially infected and infected cases, it is probable that this series consisted entirely of potentially infected cases. Nevertheless, the mortality was less than that in cases in which the classic technic was used. However, the Latzko operation was always difficult and rarely presented an adequate space through which the baby could be delivered, and tears of the peritoneum and injuries to the ureters, bladder and uterine vessels frequently occurred.

The modern concept of the extraperitoneal approach lies in the principle first expounded by Physick of Philadelphia to DeWees[68] in Horner's letter in 1824. This supravesical approach was not attempted until Frank[69] and later Sellheim[70] unsuccessfully tried it. The same approach is the basis for a series of operations presented by Waters[58] in January, 1939, with one radical difference: the plane of dissection. Previous operators had attempted to peel the peritoneum from the bladder and pelvic fascia, and Waters showed that the proper technic was to lift the peritoneofascial flap as a unit. Ricci and Marr,[71] in an excellent monograph, present a clear description of the anatomy of the area, as well as a detailed discussion of their

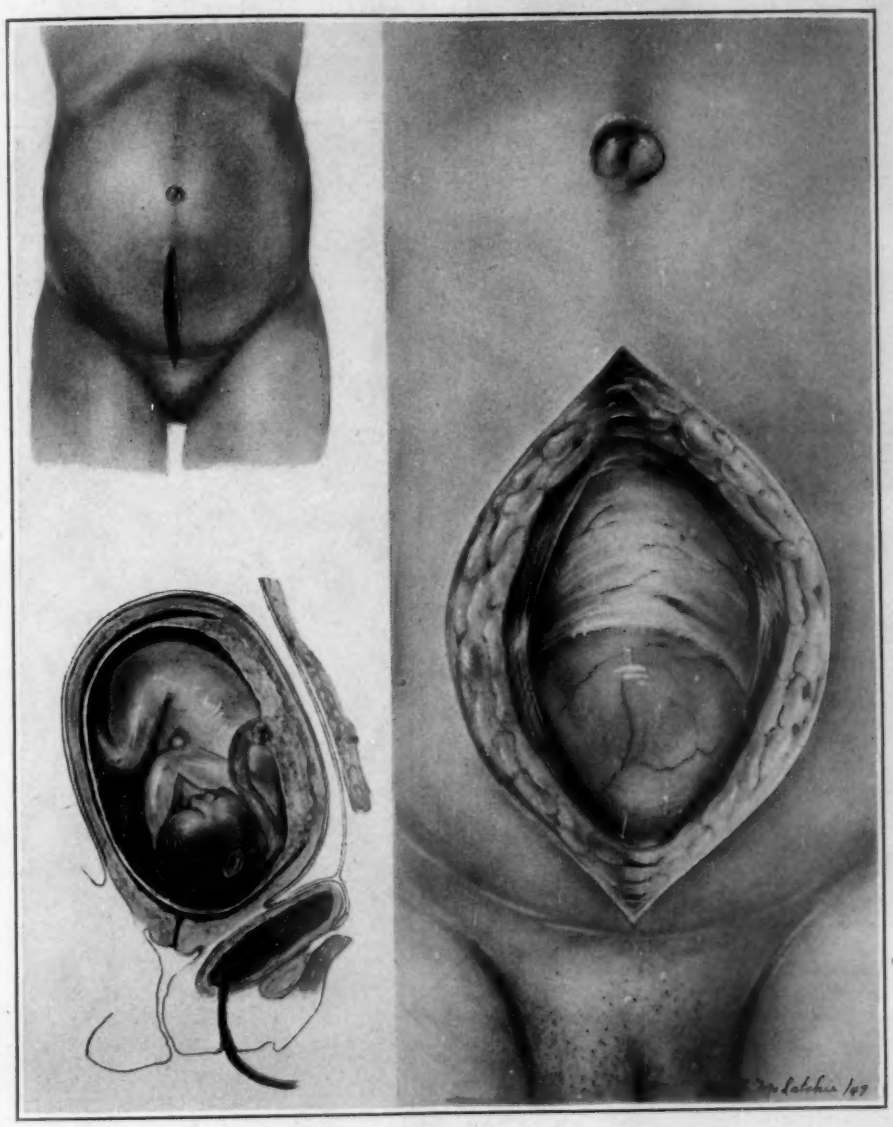

FIGURE 1.

The upper left illustration demonstrates a low suprapubic incision (extending from 5 cm. below the umbilicus to the pubis).
The lower left illustration shows a sagittal view (the double lines indicate peritoneal reflections).
The right illustration demonstrates an incision to the transversalis fascia, showing separated recti and a distended bladder.

method. Norton[72] describes a procedure that lies between the old Latzko operation and the methods of Ricci and Marr and Waters, in that the bladder is not completely separated from the peritoneal fold, the approach being paravesical and the displacement medial. The approach today involves either the complete or incomplete dislocation of the bladder from its lower-segment bed, its displacement downward or medially after complete or incomplete separation from the peritoneal fold, resulting in an adequate extraperitoneal approach to the lower uterine segment. Since the first presentation of this modern method we have been able to collect from the literature a series of 1089 cases of extraperitoneal cesarean section with a mortality of 0.8 per cent (Table 4). It is important to note that there were deaths from sepsis. It is clear, then, that this operation is not offered as a cure for infection, but rather to prevent the spread of actual or potential infection and to minimize its effect by keeping it localized to the uterus. Naturally, the antibiotics and chemotherapy are employed when indicated.

FIGURE 2. *Right Rectus Muscle Retracted Laterally, Exposing the Paravesical, Yellow, Chicken Fat Pad, Lateral to the Base of the Bladder.*

In 1942 we began to use this operation in actually or potentially infected patients, and we have been so impressed with its value that we frequently employ it now in clean, elective cases. Figures 1 to 8 outline the salient features of the technic.

Although any anesthetic may be used, we have preferred small doses of spinal anesthesia — the danger of doses of average size in obstetrics is too little realized, and we urge the selection not only of a competent anesthetist but also of a minimal dose of the drugs employed. We use 50 mg. of novocain crystals and 5 mg. of pontocaine in 2 cc. of spinal fluid. Injection is made in the fourth interspace without barbiturates, unless the operation is prolonged for any reason, when a small amount of

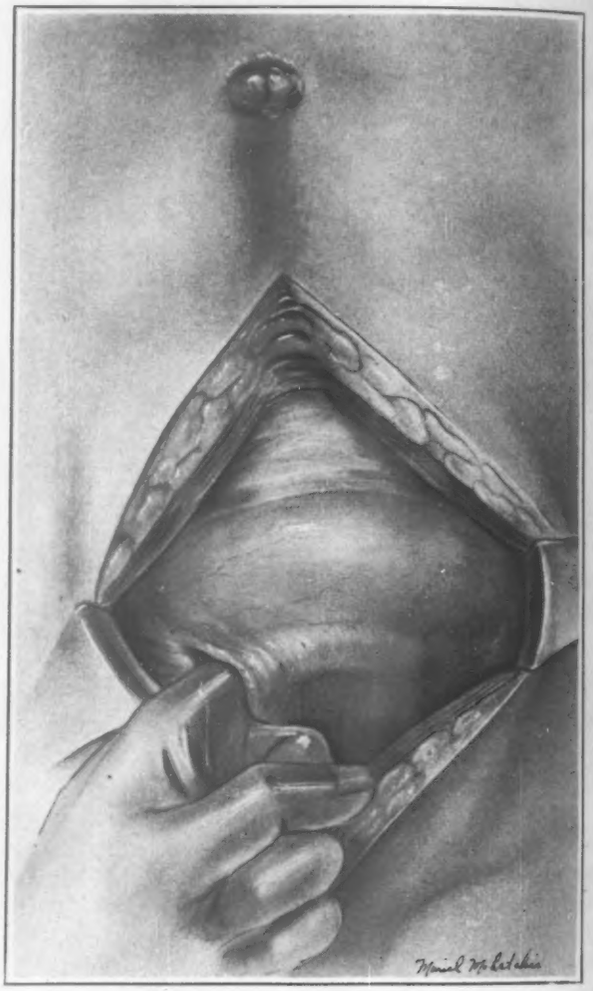

FIGURE 3. *Paravesical Fascia and Fat Pad Separated and Finger Inserted between the Posterior Surface of the Bladder and the Lower Uterine Segment (the Procedure is Repeated on the Other Side).*

intravenous barbiturate or inhalation anesthesia may be used as a supplement.

We have made use of this operation in 72 cases to date, and in our experience there has been no fetal or maternal mortality. The most impressive feature, however, has been the complete absence of postoperative morbidity. Such patients may eat within six hours of operation and have no nausea, no vomiting or "gas pains" and no more distention than occurs after a normal delivery from below. When indicated ambulation may be permitted as soon as the effect of the anesthesia has subsided.

As we grew familiar with this technic and realized through experience that the operation itself presented no hazard to the mother we gradually

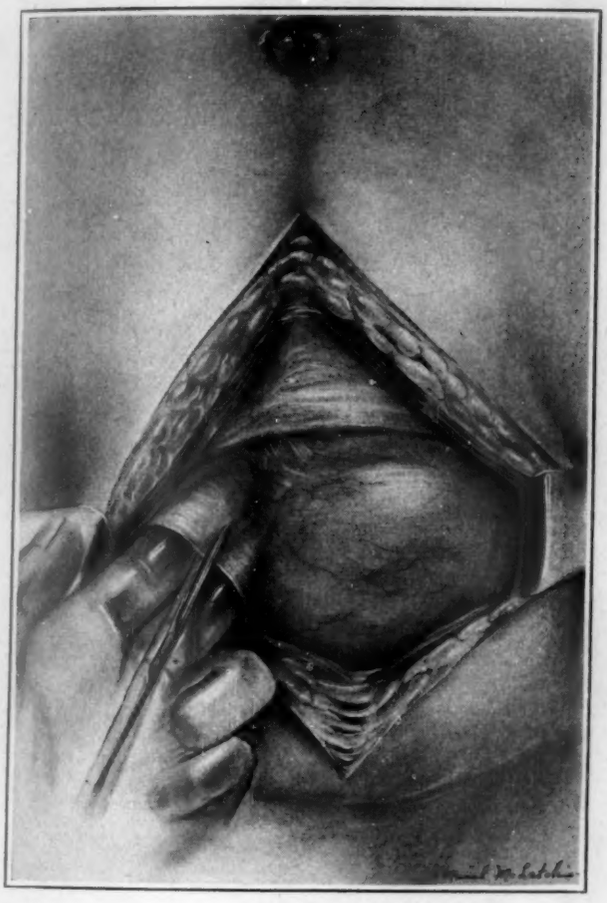

FIGURE 4. *Incision Started through the Transversalis Fascia, below the Peritoneal Reflection.*

widened our scope of indications, and at the present time we use it for the following conditions:

Infection. We consider the operation indicated in any patient in whom there is the slightest suspicion of infection, either genital or extragenital. Dehydration fever can easily be diagnosed and eliminated by proper treatment.

Ruptured membranes. The procedure is indicated in any case in which the membranes have been ruptured for a period of eight hours. Although this duration is arbitrary amniotic infection may be present in an early stage without systemic reaction.

FIGURE 5. *Lower Uterine Segment Exposed — Peritoneofascial Fold Retracted Upward, Bladder Mobilized and Drawn Downward.*

The sagittal view shows the lower uterine segment exposed extraperitoneally. The double line indicates the peritoneal fold, now completely separated from the bladder.

Labor. Actual infection of the uterine cavity potentially starts with the onset of labor, and there is no method of absolutely eliminating cases that are free of infection. Any patient in labor for six hours or longer should have the added insurance of this procedure. Harris and Brown[82] found no sterile cultures of the lower uterine segment at operation when labor had lasted six hours or more.

Vaginal examination. The operation is performed in patients who have had vaginal examinations or repeated rectal examinations.

Unsuccessful trial at pelvic delivery. When it is found that an attempted forceps operation can be concluded only with serious damage to the mother or the baby, or both, the forceps should be removed, and an extraperitoneal cesarean section performed. Irving[25] has shown that at the Boston Lying-in Hospital a difficult mid-forceps operation can be almost as dangerous to the mother as an intraperitoneal cesarean section — and about eight times more dangerous to the baby.

FIGURE 6.

Transverse incision in the thin layer of the anterior uterine fascia permits the peritoneofascial fold to be displaced cephalad. This allows adequate exposure to the lower uterine segment, even though the patient has not been in labor. The bladder has dropped beneath the symphysis-Doyen retractor shown in position.

Previous phlebitis or extensive varicosities. Earlier ambulation after the extraperitoneal operation is a definite advantage in the patient with a history of previous phlebitis and extensive varicosities.

Poor surgical risks. This category includes all patients generally regarded as poor surgical risks — that is, those with chronic respiratory infection or severe pre-eclampsia. The correlation between blood loss and sepsis is well known, and we have done this operation in placenta previa and separated placenta with this thought in mind. Patients suspected of poor healing qualities, such as those suffering from malnutrition or avitaminosis, are best protected by the extraperitoneal approach to prevent wound dehiscence and evisceration with its attendant high mortality — an event occurring in about 1 in 600 cases.[25]

Patients at or near term, when the lower uterine segment is well developed, are the best subjects for this operation. We have done the operation as early as thirty-one weeks. It can be performed with ease on patients not in labor. Drainage is performed only in patients believed to be actually infected, a "fish-tail" drain being applied to the uterine incision; this is withdrawn on the fifth day. The bladder is placed on constant drainage for a period of twenty-four hours, after which all patients have been able to void spontaneously without retention.

FIGURE 7. *Curved Incision of Lower Uterine Segment Started, Exposing Amniotic Sac.*

Note the pronounced convexity.

SUMMARY

Although better obstetric management, improved operative technic, replacement of blood loss and the

advent of chemotherapy and the antibiotics have considerably lowered the incidence of peritonitis after cesarean section, the complication still occurs, especially after the classic procedure. This type of abdominal delivery has so many disadvantages that it should be used only when better methods are not applicable or when the uterus is to be removed.

The lower-segment operation is a very satisfactory type of abdominal delivery in uncomplicated, clean cases, and although over two times safer than the classic, it is not without danger in the potentially infected case, since it does not ensure protection against peritonitis from the spill or seepage of infected uterine contents.

Craniotomy on the living child is never justifiable.

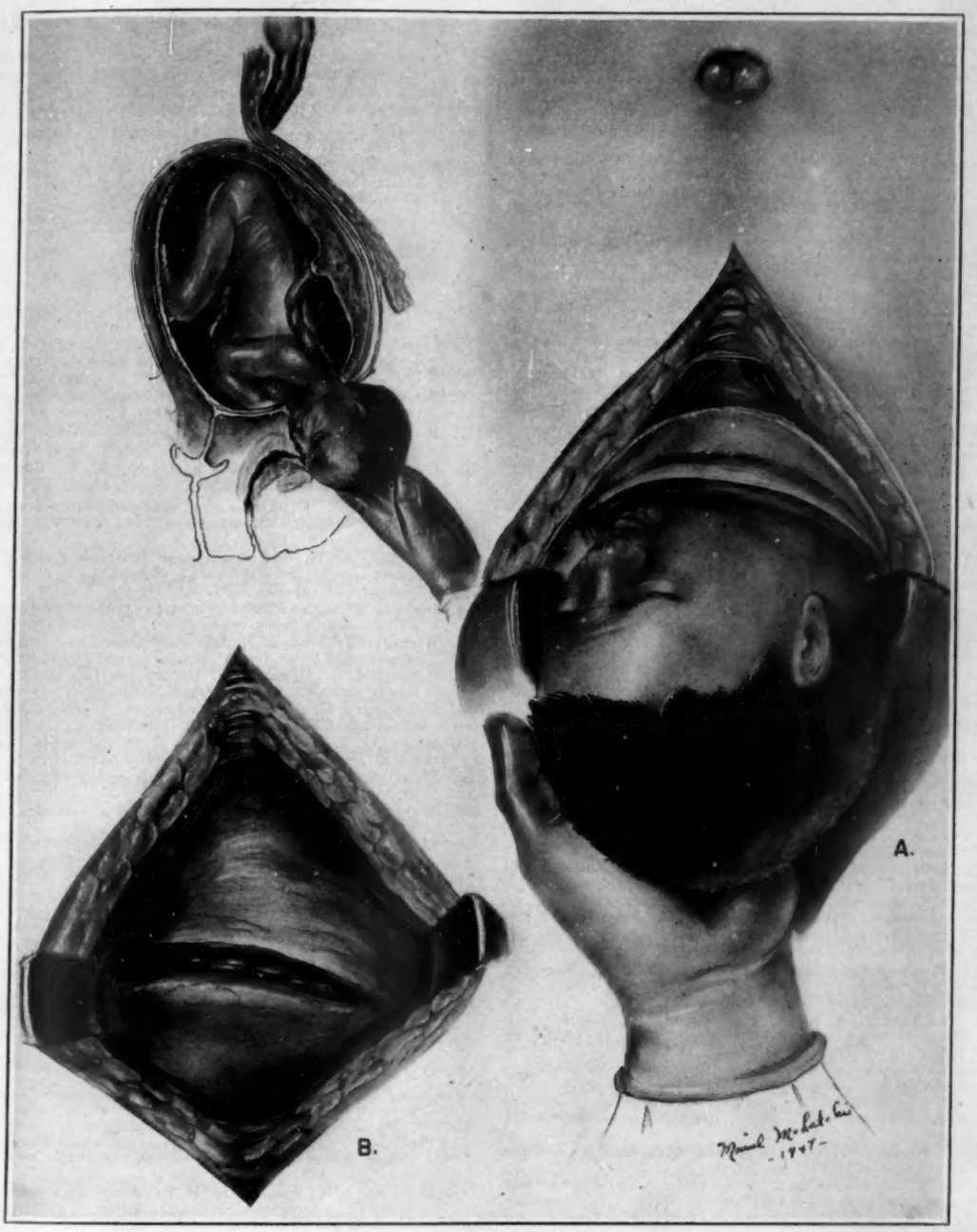

FIGURE 8. *Sagittal View, Demonstrating Extraperitoneal Extraction Effected by the Use of the Fingers as a Vectis, Assisted by Suprafundic Pressure.*

A *shows a similar anterior view.*

B *shows the uterine incision closed and the bladder refilled to check integrity.*

Cesarean hysterectomy should be used only when some pathologic condition necessitates the removal of the uterus.

The modern extraperitoneal operation not only is the safest technic for the infected or potentially

infected parturient patient but also may be used to advantage for less imperative indications.

1101 Beacon Street

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THE WORK OF A PHYSICAL-FITNESS CLINIC

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THIS is a report of a physical-fitness clinic conducted at the Massachusetts headquarters of the Blue Cross among approximately 900 executives and employees.

The personnel conducting the clinic consisted of one experienced physical-fitness worker; three physicians, part time, especially trained to make physical examinations to detect not only disease but also any physical defects that might cause impaired health; and a secretary trained in statistics, to tabulate results and to act as receptionist and make appointments. I supervised the work and saw problem cases one morning each week.

The physical-fitness worker and the secretary were on full time. One of the three part-time physicians worked six hours a day for one day and three hours a day for four days each week.

PROCEDURE

Every applicant to the clinic was given a health examination, with the object of identifying the causes of impaired health. This examination was uniform, over a hundred factors relating to the causes of health impairment being checked.

The examination consisted of a health history of the applicant, a check-up of twenty of the most common faulty health habits, a check-up of the daily program for twenty-four hours in a typical day, a check-up of all food taken in a twenty-four-hour period and a physical examination to detect not only actual disease but also any physical defects that might impair health.

The time given to each health examination and directions for the correction of both physical defects and faulty health habits was exactly sixty-five minutes. Two applicants were examined each hour, or 36 a week. The remaining time of the physical-fitness worker was devoted to return visits, especially to those who were in the danger zones of overweight and underweight. Because of the relation of weight to mortality, recognized in insurance work by rejection or substandard ratings on this factor alone, the group was separated into three divisions: overweight, optimum weight and underweight.

The optimum weight zone was determined by the range of lowest mortality found in the *Medico-Actuarial Table* of 1912 (Fig. 1). This zone extends approximately from 5 to 15 per cent above average weight and closely corresponds with that determined by clinical standards and individual health experience. Overweight includes those above optimum weight, and underweight those below optimum weight.

WEIGHT STATUS

In a period of approximately six months, 735 executives and employees, seventeen to sixty-three years of age, received this health service. The findings are presented in Table 1.

FAULTY HEALTH HABITS

Health habits that deprived the subject of one or more of the essentials of health — namely, fresh air and sunlight, proper food and food habits, regular exercise and proper rest — were considered faulty. To these must be added regularity of living habits, which is a fundamental factor in acquiring physical fitness.

The faulty health habits found by a check-up on each applicant are presented in Table 2.

The faulty health habits found in each weight zone are presented in Table 3, which demonstrates that applicants of normal weight had fewer faulty health habits than either the overweight or the underweight subjects. The most frequent faulty habits of the overweight applicants were no regular rest periods, insufficient exercise and outdoor sunlight, fast eating, habitual overeating and candy between meals. Those of the underweight group were no regular rest periods, insufficient exercise and outdoor sunlight, fast eating, candy between meals and irregular bedtime.

PHYSICAL DEFECTS

The physical defects found are presented in Table 4. Each employee was referred to his own physician or dentist for defects needing correction. Thus, 197 (27 per cent) applicants were referred to family physicians, and 124 (17 per cent) to family dentists.

When his faulty health habits had been checked and the physical examination completed, the applicant was told what health habits and what physical defects were found, and their effect in impairing his health was explained. He was also told that both overweight and underweight were unnecessary and that by correcting his habits that were essentially faulty he could improve his health and thus add years to his period of active work and efficiency.

He was then asked this question. "Are you interested in planning your day's activities to correct these defects and to work for better health?" If he said he was interested, it was suggested that he weigh in each week, and he was told that he would be helped to plan his daily program to attain better health.

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Return Visits

After the examination had been completed, the applicant was offered an opportunity to return each week for suggestions regarding the most efficient way to gain or lose weight and how to make changes in his daily program of activities to correct faulty health habits and secure the essentials of health.

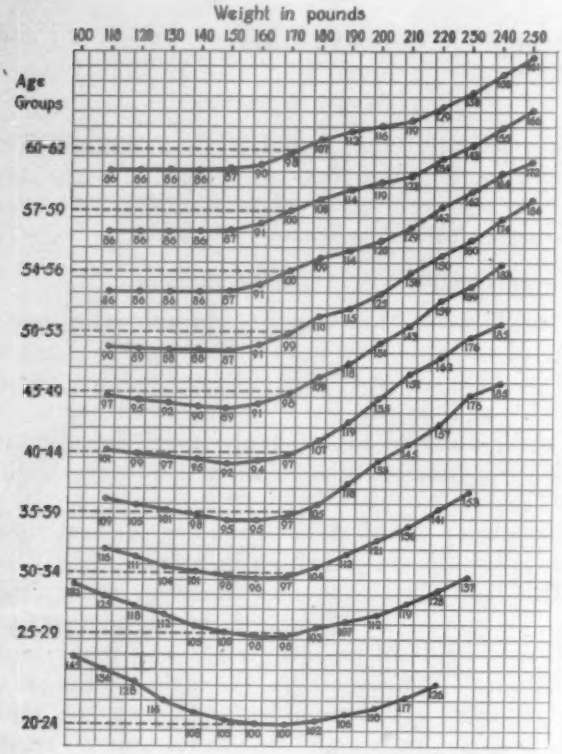

Figure 1. *Influence of Weight on Mortality.*

The figures on the curves represent the percentage of mortality for the respective ages and weights. In the early twenties mortality increases about 1 or 2 per cent below average weight for height. Above the age of thirty-five mortality increases a like percentage for each pound above optimum weight (Reproduced from Emerson[1]).

Table 1. *Weights of 735 Men and Women.*

Status of Subjects	Range	No. of Cases	Per-centage
	lb.		
186 men:			
Overweight	2–64	46	25
Normal weight	—	50	27
Underweight	1–35	90	48
549 women:			
Overweight	1–103	118	22
Normal weight	—	189	34
Underweight	1–34	242	44
735 men and women:			
Overweight	1–103	164	22
Normal weight	—	239	33
Underweight	1–35	332	45

In answer to the question, "Have you any health habits that are injurious to your health," 730 of the 735 applicants taking the service answered "No." Yet, when their daily programs were checked up, a total of 2294 faulty health habits were found — an average of 3.1 habits that needed to be corrected.

The response to the offer of an opportunity for return visits was so great that soon it became necessary to reduce the number of initial examinations to take care of the return visits. In the limited time of the demonstration, the number of return visits made by those interested in working for a higher standard of health was 2822.

Table 2. *Faulty Health Habits (All Applicants).*

Faulty Health Habits	No. of Cases	Per-centage
No regular rest periods	717	98
Insufficient exercise or outdoor sunlight	273	37
Fast eating or washing down food	206	28
Candy or sweets between meals	165	22
Worry and fretfulness	143	19
Removable physical defects uncorrected	128	17
Habitual overeating or undereating	114	16
Irregular bedtime	112	15
Irregular time of bowel movement	105	14
Irregular habits of living	71	10
Excessive use of tea, coffee, alcohol or tobacco	59	8
Excessive fastidiousness about food	47	6
Sleeping with windows closed	41	6
Overdoing at work or play	39	5
Irregular mealtimes	32	4
Eating when overtired	19	3
Inadequate vacations or weekly rest	10	1
Uncontrolled likes and dislikes	8	1
Habits injurious to health	5	1
Working in poor air (above 68°)	0	0
Total	2294	
Average		3.1

At each return visit, the applicant was weighed and his diet list checked, and suggestions were made for rearranging his daily program to ensure him essentials of health.

Table 3.. *Faulty Health Habits According to Weight of Applicant.*

Status of Applicants	No. of Cases	Faulty Health Habits	
		No. of cases	Average
Overweight	164	559	3.40
Normal weight	239	687	2.87
Underweight	332	1048	3.15
Totals	735	2294	
Average			3.10

The time given for each return visit from the time of entering the clinic to the time of leaving was an average of eight minutes.

In this demonstration, as elsewhere, the return visits were found to be of great importance, because habits of daily living are determined not by the requirements of healthful living but by individual family life, working conditions, social life and diver-

sions that, from the standpoint of health, may be dissipations.

Directions for improving health were given by the physician and physical-fitness worker together, on the basis of the findings recorded on the examination form. Directions were given as definitely as those given by the physician after he makes a diagnosis of disease.

The success of the applicant in gaining or losing weight, in reducing his blood pressure and in increased efficiency and improvement in general well-being kept him returning to the clinic until his objective had been attained. He was made to understand that the job was his, and that better health could be won only by his own efforts.

Results

Overweight Applicants

It was suggested to the overweight applicants that they reduce their weight slowly, the reduction not to exceed an average of 1 or 2 pounds a week, until they got out of the danger zone of overweight indicated in Figure 1 — the men, until the abdomen ceased to be prominent and the women, until the folds of fat disappeared from about the waist and hips.

The weight reduction accomplished in the limited time of the demonstration varied from 2 to 27 pounds, a total of 574 pounds.

The following case provides an example:

J. M., a 19-year-old girl, had always been overweight and resented being called fat. She had tried various methods of reducing her weight without success. By reducing her diet (calories) to a point where she lost about 1.5 pounds a week, she reduced her weight 27.5 pounds. During that time, she did not suffer from hunger after the 1st week and considered the diet that met her needs no special hardship.

Underweight Applicants

The underweight applicants were found to have more faulty health habits than either of the other groups. Their daily programs showed almost continuous nervous tension, caused not so much by the pressure of work as by continuous activities outside working hours and on Saturdays and Sundays.

Their gain in weight during the time of the demonstration ranged from 2 to 14 pounds, a total of 246 pounds.

An example is afforded by the following case:

E. C., a 19-year-old girl, was attractive and ambitious. She was working overtime 1 or 2 nights a week and studying designing 2 nights a week. She had no physical defects. Her faulty health habits were those of taking no rest periods during the long day and taking a light breakfast and a light lunch, and eating a hearty dinner at night when overtired.

She was advised to take a complete rest, lying down for 20 minutes before her evening meal, and on Saturdays and Sundays to have her breakfast at the regular time, resting in bed until noon and utilizing the afternoon for outdoor exercise and sunlight. She was able to continue night and overtime work and gained 14 pounds — attaining normal weight for her height.

Hypertensive Applicants

There were 12 cases of hypertension, the blood pressure ranging from 150 to 210 systolic and from 90 to 110 diastolic.

By loss of excessive weight and relief of high-pressure living by twenty-minute periods of complete rest before the evening meal, and by correction of faulty health habits, the reduction in systolic blood pressure averaged 20.

Table 4. *Physical Defects Discovered.*

Defect	No. of Cases
Underweight	332
Overweight	164
Pronated feet	154
Carious teeth	139
Nasopharyngeal obstruction	73
Spinal curvature	24
Acne	18
Malocclusion	12
Hernia	6
Varicose veins	4
Nasal polypus	3
Bronchitis	3
Furunculosis	2
Dermatitis	2
Psoriasis	2
Scabies	2
Fungous inflammation of feet	2
Sinusitis	2
Chronic otitis media	2
Conjunctivitis	2
Hyperthyroidism	2
Mastitis	2
Hemorrhoids	2
Varices	2
Pilonidal cyst	2
Ringworm	1
Keratitis	1
Pterygium	1
Blepharitis	1
Goiter	1
Arrested tuberculosis	1
Perforated eardrum	1
Hay fever	1
Trench mouth	1
Appendicitis	1
Stomatitis	1
Valvular heart disease	1
Rheumatoid arthritis	1
Spinal arthritis	1
Total	972
Average	1.32

The faulty health habits in these cases were those of habitual nervous tension, insufficient exercise and outdoor sunlight and fast eating.

The following cases are illustrative:

H. M., a 56-year-old man, weighed 191 pounds. He had recovered from a heart attack but was short of breath on slight exertion. His heart area was enlarged, and a systolic heart murmur was heard over the entire pericardium. His faulty health habits were habitual overeating, worry, no rest periods and the candy habit.

He reduced his weight 13 pounds and his systolic blood pressure from 210 to 146. He was able to continue his regular work without untoward symptoms. In the meantime he was referred to his own physician for a more careful supervision of his heart condition.

D. R., a 50-year-old woman, weighed 159 pounds. Her blood pressure was 150/50. She reduced her weight 25 pounds, and her blood pressure returned to the normal of 116/82.

These findings and results closely parallel those observed in a group of 100 consecutive cases of hypertension reported in September, 1938.[2]

Absenteeism

All executives and employees were circularized before the work began. Therefore, an adequate control group was not available to compare days off from sickness.

However, a study of 100 employees taking the service and of 100 before taking the service (matched by five-year age ranges, by sex — 26 women and 24 men — and to some extent by employment status) showed a reduction of 26 per cent in days off from sickness for those taking the service,[3] as compared with 46 per cent in a large group elsewhere[4] when controls were complete.

Discussion

Because of the loss to industry caused by absenteeism, and because of the greater loss in lessened efficiency of the workers when on duty, it is only logical that definite steps should be taken to prevent sickness. The success of the physical-fitness service in accomplishing this purpose, wherever tried, has demonstrated its value.

The cost of the service is relatively small because the bulk of the work is done by a lay person, the trained physical-fitness worker. One such person, with the part-time aid of a physician and a secretary, is able to conduct the service for a group of from 1800 to 2000 persons and handle from 4000 to 5000 return visits a year.

Patients are willing to pay for medical service when they are sick but not for health service when they think they are well. Therefore, the expense must be borne by the employer. The employer is justified in assuming this expense because of resulting reduction in absenteeism, increased efficiency and improvement in morale.

To the cost of days off for sickness must be added the loss from lessened efficiency, for when health is impaired the higher powers are first affected — judgment, initiative, endurance and personality. This loss is most evident among the higher paid executives, on whose efficiency the success of the industry rests.

True health examinations, which are necessary for successful health work, are not now available because the usual so-called "health examination" is an examination for disease. Thus, to obtain its benefits, the subject must be sick before he can be well.

The physical-fitness clinic functions as a screening clinic not only to determine the causes of impaired health but also to identify disease and send such patients to their own physicians and dentists.

The number of physical defects and faulty health habits and the number of return visits of the female employees were in like proportion to those of the males.

The twenty-four-hour list of activities demonstrates the helplessness of the average person in planning his day to conform with the essentials of health. The fault lies not so much in working conditions, in the improvement of which industry has made epochal advances, but chiefly in the time off from work — especially Saturdays and Sundays, which are usually periods of irregularity and of activities that make for dissipation of health reserves, rather than of recuperation. Hence, "Black Monday" is proverbial in industry.

By his return visits to the clinic, the applicant is encouraged and receives the fundamental health instruction necessary for him to obtain optimum results. During the period when he is working for better health by correcting his faulty health habits, he forms good health habits that continue throughout his working life, for good habits are as persistent as faulty ones.

The physical-fitness clinic, properly conducted, brings to the patient not only the resources of present-day medical knowledge but also the newer knowledge of factors contributed by allied science — especially those relating to the functions of the body as affected by irregularity, overfatigue, lack of sunlight and disturbed physiologic processes.

The diagnosis of health is a challenge to the best skill of the physician — as stated by Theobald Smith, "The diagnosis of health is more difficult than the diagnosis of any disease."

The desire for better health is universal, and the patient needs only to be convinced that habits are really faulty, and to receive definite directions for correcting them, to co-operate willingly. His success makes the atmosphere of the clinic a happy one both for himself and for the clinic workers — all striving for a common purpose.

Summary and Conclusions

A physical-fitness service offered to approximately 900 executives and employees of the Blue Cross at their Boston home office is described.

The results obtained answered wholly or in part the following questions:

To what extent were they interested in working to improve their health?

Attendance at the clinic was voluntary except for 313 new employees who entered the organization with the understanding that they would be required to have the health examination and return once a year for a check-up; 422 of the remaining old employees applied for the examination — a total of 735.

Did they know they had health habits essentially faulty?

Only 5 applicants answered in the affirmative when asked, "Have you any health habits injurious to your health?" Thus 730 were unaware that they had such faulty health habits, although when checked 2294 were found.

When faulty health habits are identified, will they correct them?

In the six-month period, 2822 return visits were made by those working to correct them.

If they work to correct these habits, what results can they obtain?

Their improved health was the cause of the continued visits. A partially controlled group of 100 applicants showed a reduction of days off from sickness of 26 per cent, as compared to a reduction of 46 per cent in a similar group in which controls were complete.

Eighty-eight cases in the danger zone of overweight lost from 1 to 28 pounds, a total of 574 pounds. Eighty-eight cases in the danger zone of underweight gained from 1 to 15 pounds, a total of 247 pounds, and 14 patients with systolic pressures ranging from 150 to 210, were found, at the end of the period of return visits, to have reduced the systolic pressure an average of 20.

Of the 735 applicants examined, 110 had no physical defects aside from overweight and underweight. None were entirely free from faulty health habits.

Twenty-seven per cent were referred to their family physicians, and 17 per cent to their family dentists.

A check-up of their twenty-four-hour periods of activity showed that faulty health habits were occasioned chiefly by conditions outside rather than during working hours.

The clinic demonstrated that both executives and employees need only to have their faulty habits identified and definite directions given for their correction to obtain efficient co-operation in working for better health.

I am indebted to Mr. R. F. Cahalane, executive director of the Blue Cross, for the invitation to conduct this clinic and to Mr. H. Proctor Redd, director of public relations, Mr. Harry Healy, director of personnel and Dr. Charles G. Hayden, medical director, Blue Cross, for their cordial co-operation.

Personnel of the clinic included Albert C. England, M.D., examining physician; George M. Olive, Jr., M.D., and L. Murray Shipp, M.D., examining physicians; Helen M. Sanderson, physical-fitness worker; and Martha Freedman, secretary and statistician.

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MEDIAL PTOSIS OF THE KIDNEY*

A New Renal Syndrome

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PROBABLY on more than one occasion urologists have examined patients for pain or discomfort that, because of its character and location, was thought to be of renal origin, only to find the pyelographic studies normal. In at least a few of these cases the symptoms may be due to medial ptosis of the kidney.

Caudal mobility of the kidney with the body in the vertical or upright position (ptosis of the kidney) has been known for many years as a clinical entity, with alternate periods of popularity and disrepute. This paper is not directly concerned with that subject.

Little or no attention has been given to medial or lateral mobility of the kidney, and the pyelographic demonstration of an abnormal degree of medial mobility has not, to my knowledge, been previously described. "Medial ptosis of the kidney" has been chosen as a term to describe abnormal medial mobility of that organ. With a patient lying on the left side the condition of medial ptosis of the right kidney would be shown by abnormal change of the position of the right kidney toward or beyond the vertebral column. Similarly, medial ptosis of the left kidney might be demonstrated with a patient lying on the right side.

This condition, which may be the cause of symptoms, cannot be demonstrated by the usual pyelographic technic. The diagnosis can be made, however, by means of x-ray films using anteroposterior projection with the patient in the lateral position (Fig. 1). After cystoscopy and ureteral catheterization or during intravenous pyelography the patient is placed on a litter lying on his side, with the painful side up. The patient's back is placed firmly against an upright x-ray table used for gastrointestinal or chest x-ray examination, or against a cassette, 35 by 65 cm., in front of which has been placed a portable Sieman's microgrid.‡ The x-ray tube is then focused for an anteroposterior view of the kidney region. A retrograde pyelogram is then made, with the ureteral catheter pulled down into the lower ureter before the x-ray film is taken. A routine film taken in this position during intravenous pyelography may be used instead.

To determine the normal extent of medial mobility of the right kidney with the patient lying on the left side, films were taken in the position shown in Figure 1 after pyelographic studies had been completed in a number of patients. As illustrated in Figures 2 and 3, these studies showed no medial descent of the kidney in the normal person.

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†Surgeon-in-chief for urology, Boston City Hospital.

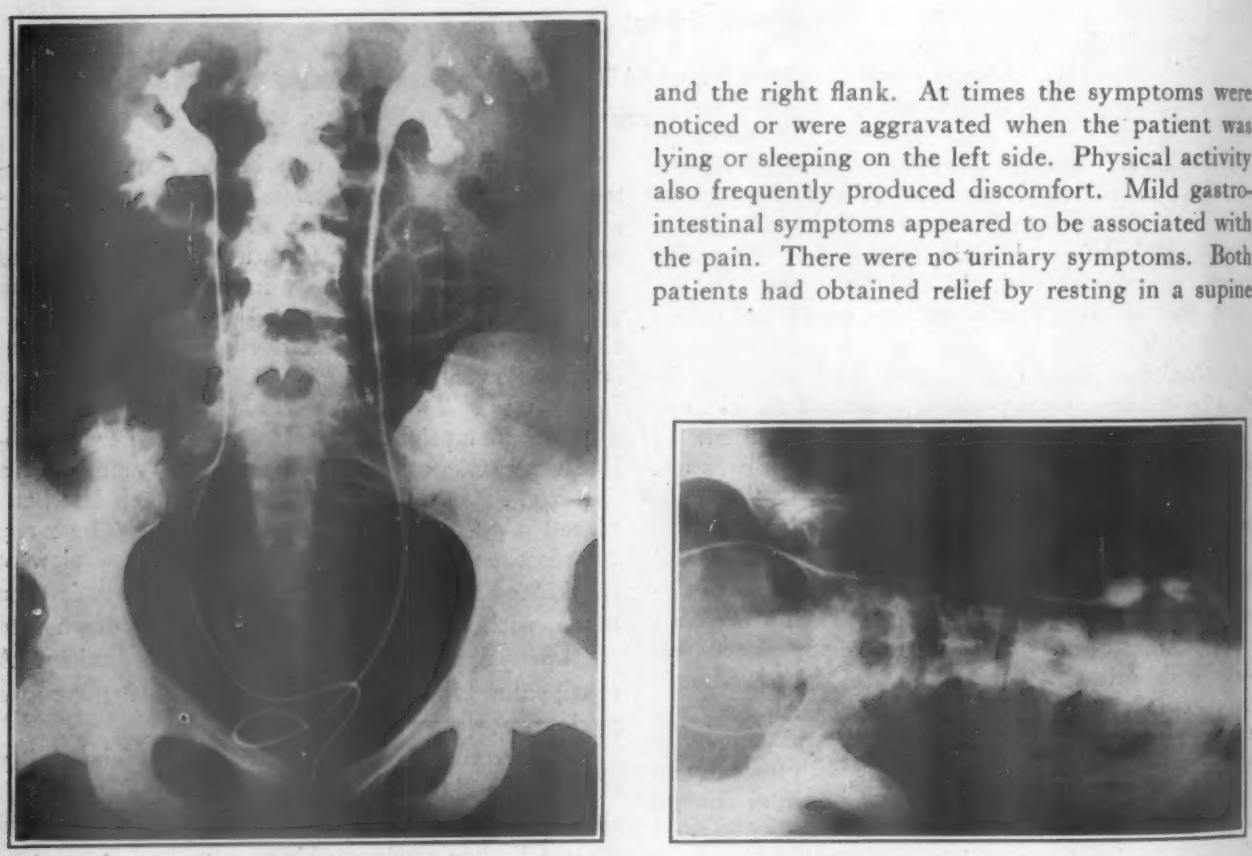
‡Obtainable from the Liebel-Flarsheim Company, Cincinnati, Ohio.

The condition of medial ptosis has recently been demonstrated and corrected in 2 patients. Both were slender women who had moderate pain and distress in the right upper quadrant of the abdomen

FIGURE 1. *Position of Patient for Pyelogram to Demonstrate Abnormal Medial Mobility of the Right Kidney.*

X-ray film taken as an anteroposterior projection, with the patient in the lateral position and with the painful kidney up.

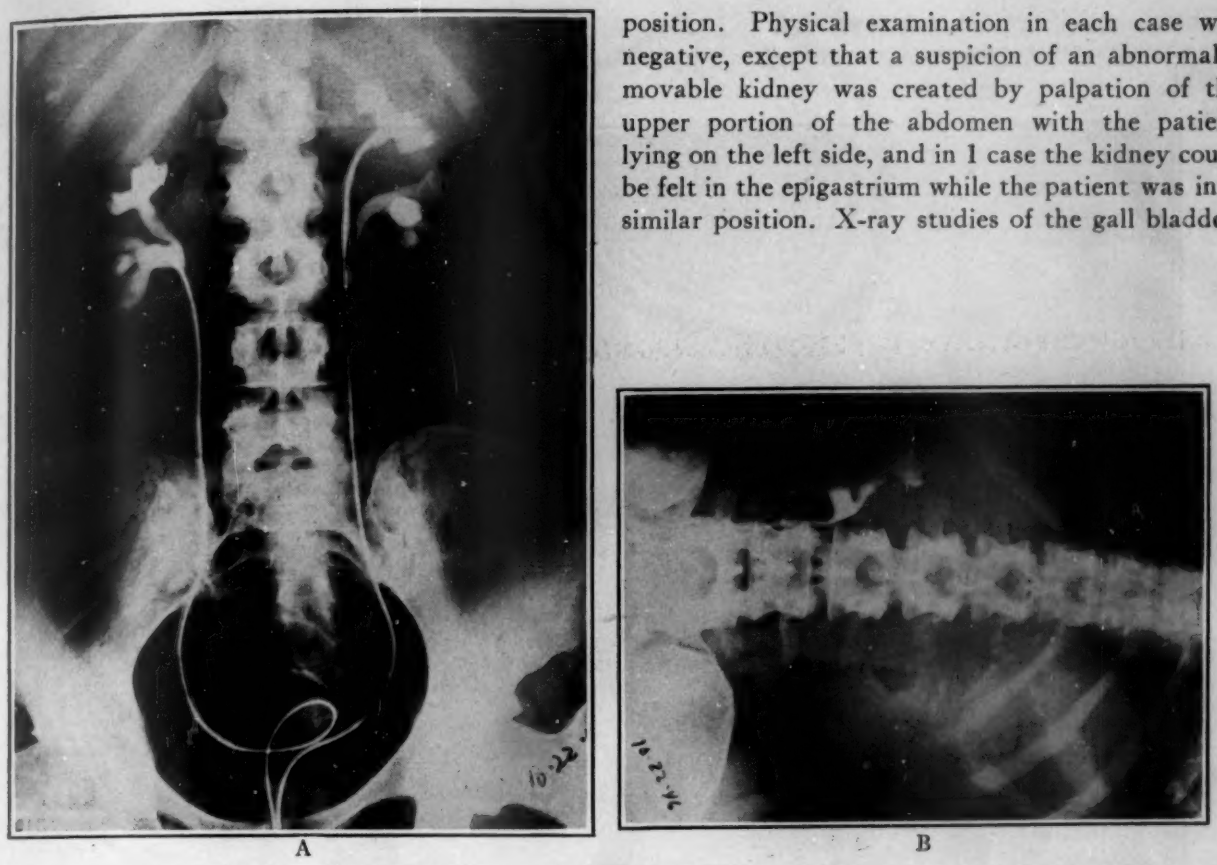
and the right flank. At times the symptoms were noticed or were aggravated when the patient was lying or sleeping on the left side. Physical activity also frequently produced discomfort. Mild gastrointestinal symptoms appeared to be associated with the pain. There were no urinary symptoms. Both patients had obtained relief by resting in a supine



A B

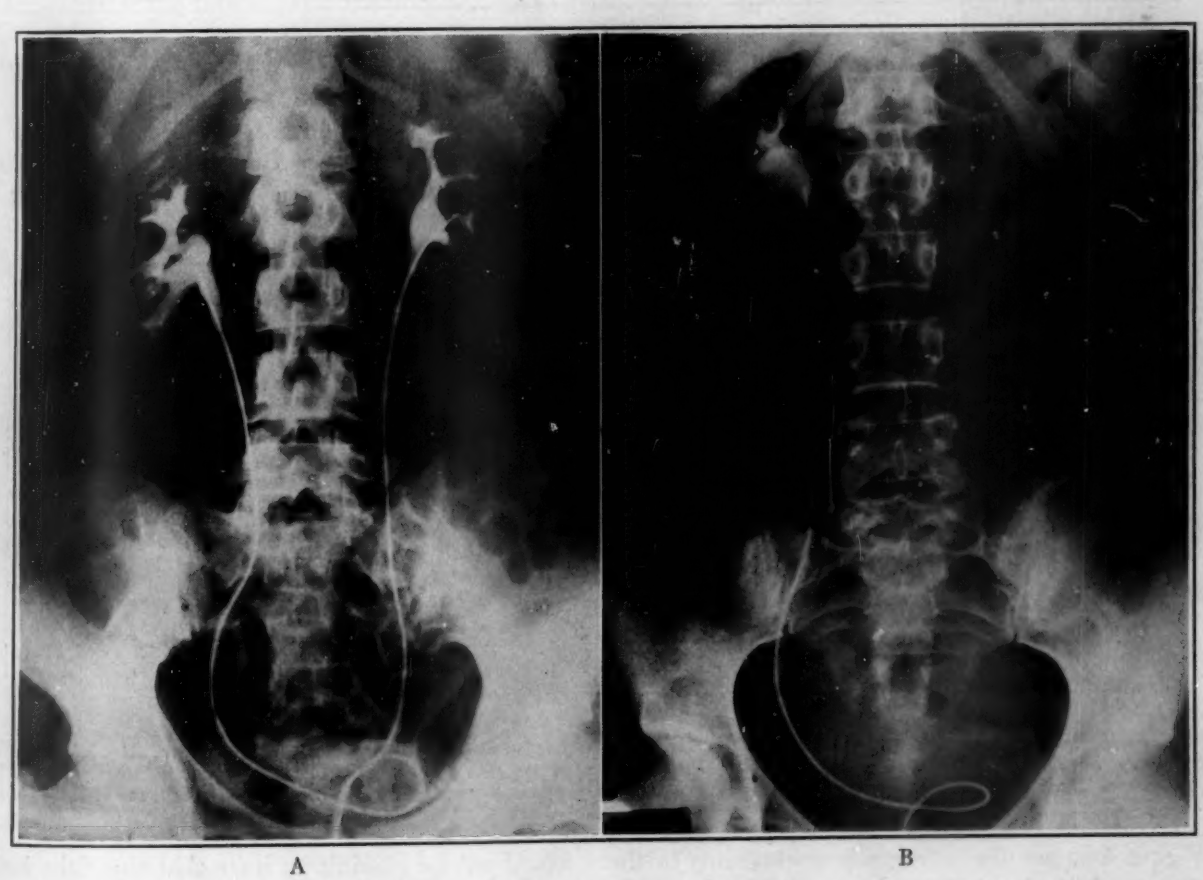
FIGURE 2. A *Shows a Normal Pyelogram in the Supine Position, and* B *a Pyelogram of the Same Patient in the Position Shown in Figure 1. (The Right Kidney Remains in Normal Position.)*

position. Physical examination in each case was negative, except that a suspicion of an abnormally movable kidney was created by palpation of the upper portion of the abdomen with the patient lying on the left side, and in 1 case the kidney could be felt in the epigastrium while the patient was in a similar position. X-ray studies of the gall bladder,



A B

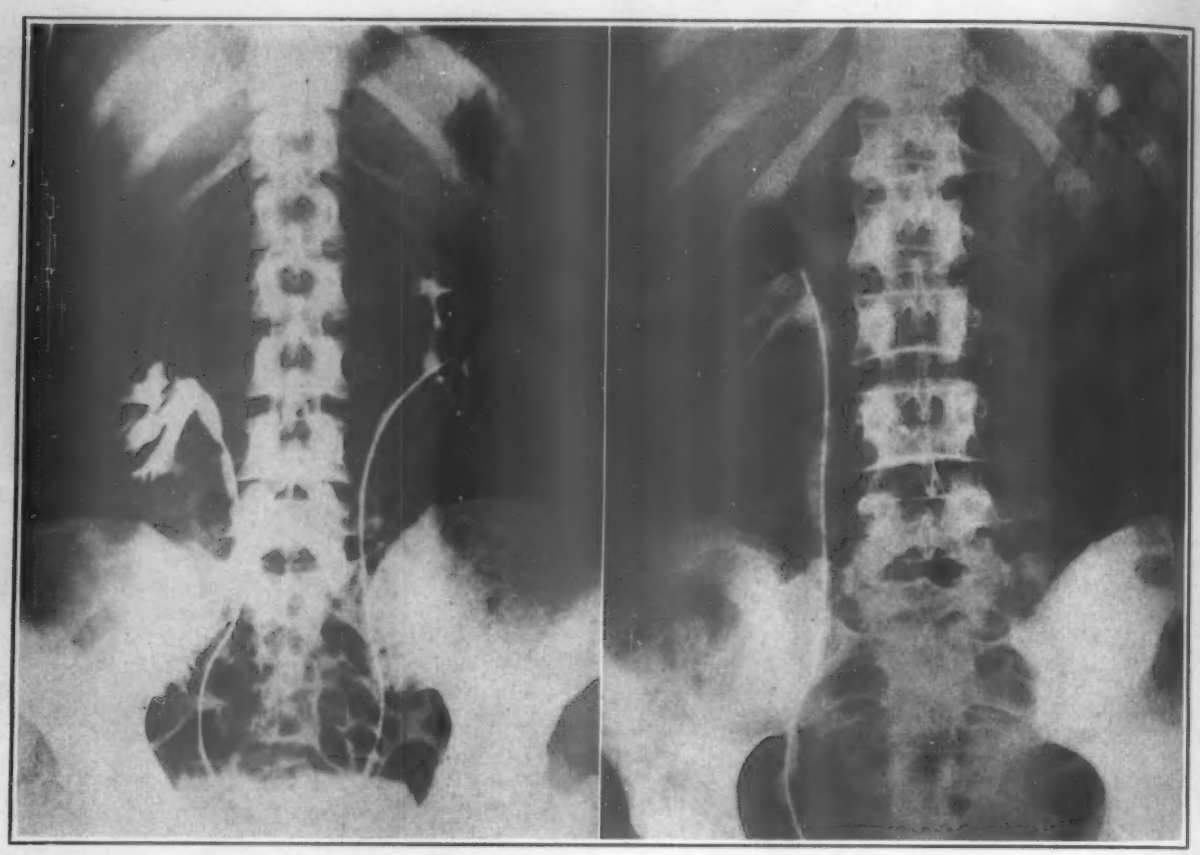
FIGURE 3. A *Shows a Normal Pyelogram in the Supine Position, and* B *a Pyelogram of the Patient in the Position Shown in Figure 1. (The Right Kidney Remains in Normal Position; Note Fluid Level in Kidney.)*



A B

FIGURE 4. A *Shows a Pyelogram of Patient "A" in the Supine Position, and* B *a Pyelogram of Patient "B" in the Supine Position.*

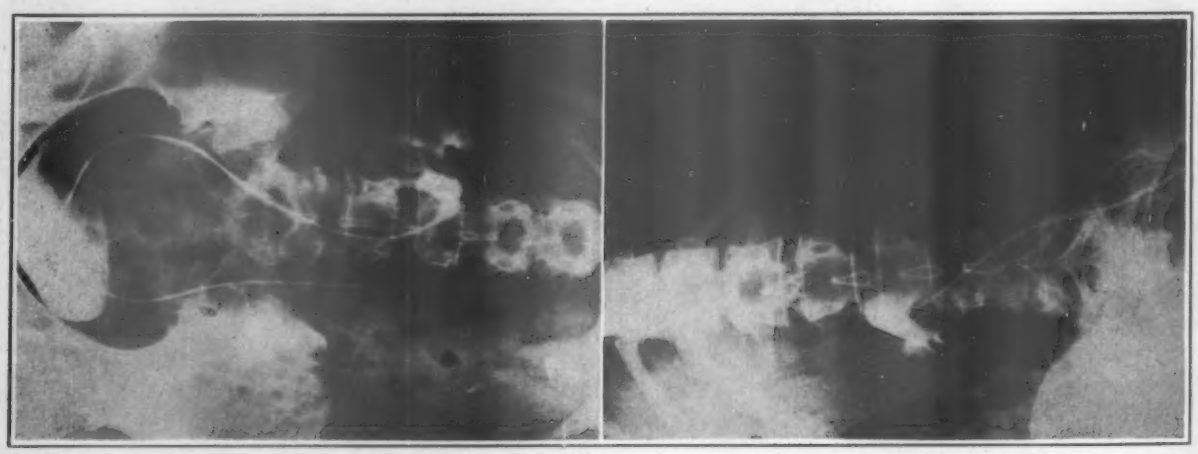
stomach and intestines were reported as negative. Intravenous urograms were likewise normal. The question of medial ptosis arose, and special studies of the right kidney established the diagnosis.



A B

FIGURE 5. A *Shows a Pyelogram of Patient "A" in the Upright Position, and* B *a Pyelogram of Patient "B" in the Upright Position.*

Figure 4 shows the retrograde pyelograms in the supine position in each patient. Figure 5 reveals the extent of caudal mobility with the patients in an upright position — patient "A" shows some, but not significant, vertical ptosis, whereas patient "B" shows none. From these films it certainly would not be possible to state that the right kidney was the cause of symptoms in either case.

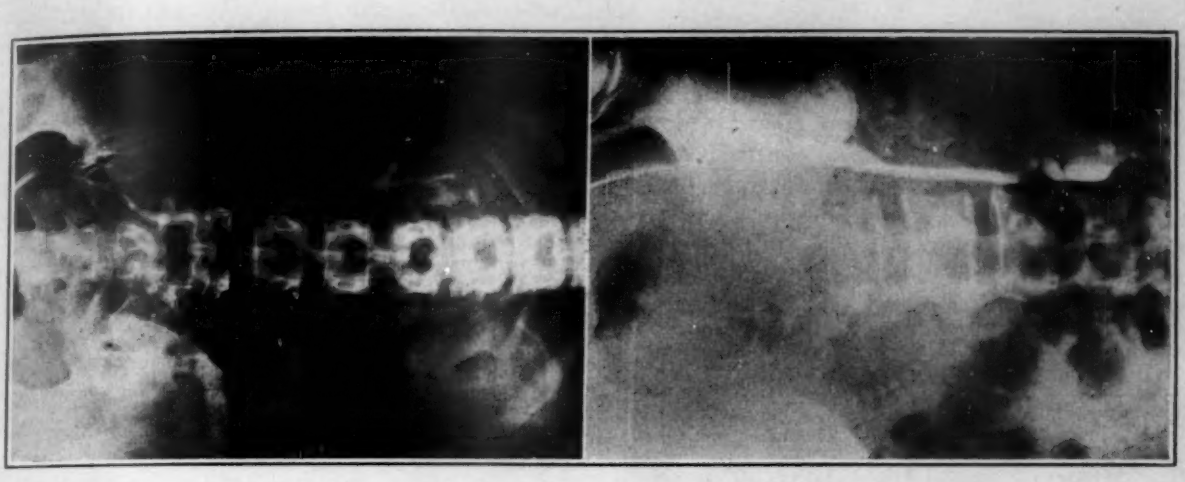


A B

FIGURE 6. A *Shows a Pyelogram of Patient "A" in the Position Shown in Figure 1 — There Is Abnormal Medial Mobility of the Kidney in This Position;* B *Demonstrates a Pyelogram of Patient "B" in the Position Shown in Figure 1 — There Is Abnormal Medial Mobility of the Kidney, Which Has Dropped Beyond the Midline and Carried the Ureter with It.*

However, by means of films taken in the position described above (Fig. 6) a significant degree of medial ptosis of the right kidney could be demonstrated in both patients; patient "B" appeared to have a considerable degree of medial mobility.

Nephropexy was performed on both patients, with complete relief of symptoms, and postoperative films in the new position (Fig. 7) showed that medial ptosis had been corrected in each case.



A B

FIGURE 7. A *Shows a Postoperative Pyelogram of Patient "A" in the New Position (Kidney Is Now in Normal Position).* B *Demonstrates by Postoperative Pyelogram of Patient "B" in the New Position, with the Kidney in Normal Position. (The Fluid Level in the Kidney and the Fetal Spine Can Be Seen.)*

It is my belief that this syndrome may explain some of the hitherto undiagnosed symptoms referable to the kidney regions and the upper abdominal quadrants. Nephropexy is a simple method of correcting the condition.

A DEVICE FOR CONTROLLED NEGATIVE PRESSURE

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BOSTON

THIS article presents a simple compact device for the safe administration of controlled negative pressure to the gastrointestinal tract of patients in institutions having built-in negative-pressure systems.

Some of the difficulties and inconveniences previously encountered in the use of suction apparatus prompted the development of this device. Any apparatus, when on the floor, is likely to be tipped over or broken and makes cleaning of the room difficult. Bulky glass apparatus, if strapped on the bed frame, is likewise often broken. Many rubber tubes leading to and from the apparatus are often draped over the bed and the patient. Glass connectors in rubber-stoppered bottles are easily shattered, and may injure the hands of the attendants.

FIGURE 1. *Photograph of the Device in Its Stainless-Steel Rack. This rack was designed and constructed by Mr. Edward Hallinan.*

This device consists of two 2-liter "Atlas" preserve jars, one for drainage and the other for overflow. The bottles are fitted with cast-brass duplicates of the original snap-on glass tops, with brass inflow and outflow tubes attached — an arrangement that allows the tops to be easily removed for emptying the bottles and to be easily replaced and made airtight. The bottles fit in a stainless-steel rack, which hangs from the frame of the hospital bed (Fig. 1). Thus, the rubber tubing is kept at a minimum, and the device is off the floor and out of the way when the bed is being made or moved.

The overflow bottle (Fig. 2) rarely needs opening except when the drainage bottle has been allowed to overflow into it. Should the overflow bottle become full, the downward extension of the outflow tube will not permit the fluid level to reach any vulnerable part. The overflow bottle has three special features. The first, a flow-control unit attached to the outflow tube, is a bolt that has been drilled with a small-caliber drill. The resulting small orifice per-

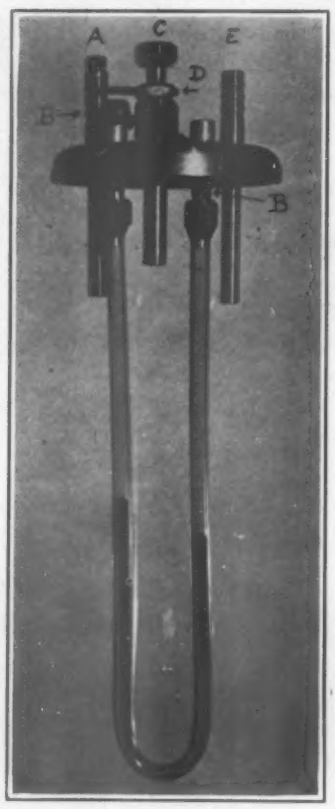

FIGURE 2. *Photograph of the Top of the Overflow Bottle, with Its Attachments.*

A *shows flow-control unit in outflow tube,* B *leather-covered manometer openings,* C *adjustment bolt for negative-pressure release valve,* D *lock nut, and* E *inflow tube.*

*Assistant resident in surgery, Massachusetts General Hospital.

mits the wall valve to be fully opened, since it automatically allows only a small volume of air to flow through the device per unit of time. Careful flow adjustments at the wall valve are therefore unnecessary. The second feature is a mercury manometer incorporated in the device, which accurately shows the observer at a glance the degree of negative pressure in the system. The ends of this manometer are leather covered. One end opens inside, and the other outside the bottle. This arrangement permits no mercury to escape and yet allows sufficient air flow for the manometer to function. The manometer tube is made of "Tenite II," an Eastman Corporation clear thermoplastic that is difficult to break. The connectors must be made of a metal such as aluminum or stainless steel that will not amalgamate with the mercury. The third feature, a negative-pressure release valve, is also incorporated in the device, with a range of adjustment from 0 to 6 inches of mercury — 3 inches of mercury is roughly equivalent to 40 inches of water, which is the usual working range for gastrointestinal suction. The negative-pressure release valve is a ball valve with adjustable spring tension. A metal ball sits on top of a spring in the hollow valve housing, and the valve seat is at the base of the hollow adjustment bolt, to which a lock nut is attached. The air intake is beneath the head of the adjustment bolt so that it cannot be inadvertently occluded.

By means of the features mentioned above a system is set up that causes a constant stream of air to flow through the overflow bottle, entering via the air intake and leaving by way of the small orifice in the flow-control unit. The negative pressure in the overflow bottle is variable, being changed by increasing or decreasing the spring tension, which forces the metal ball against the valve seat (Fig. 3). The negative pressure is registered on the manometer and transferred through the drainage bottle to the patient. Variations in the negative pressure in the main hospital system do not affect the negative pressure in this device, provided the negative pressure in the hospital system does not fall below that in the device.

Summary

A device for safely administering controlled negative pressure to the gastrointestinal tract of patients is presented. The apparatus is simple in design, and the parts are inexpensive. Its breakable parts are its cheapest and are readily obtainable. It has proved simple to operate for members of the nursing staff who have used it.

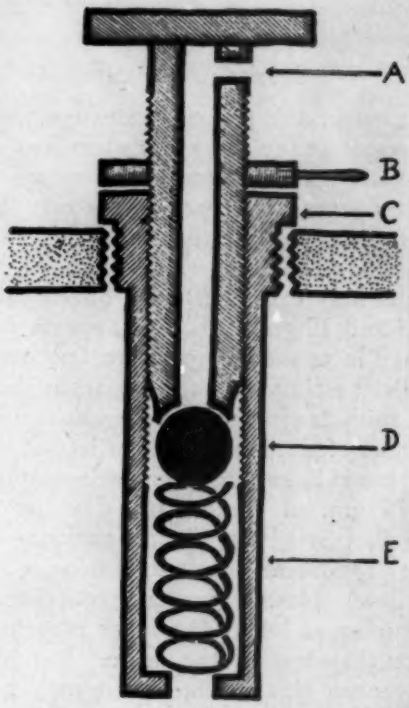

FIGURE 3. *Diagram of the Negative-Pressure Release Valve.* A *demonstrates air intake beneath the head of the adjustment bolt,* B *lock nut, with attached handle for tightening and releasing,* C, *hollow-valve housing,* D *metal ball, and* E, *spring forcing metal ball against the valve seat.*

MEDICAL PROGRESS

THE "NEPHROTIC SYNDROME" (Concluded)*

STANLEY E. BRADLEY, M.D.,† AND CORNELIUS J. TYSON, M.D.‡

NEW YORK CITY

HYPOPROTEINEMIA

The return of the plasma protein concentration toward normal as the urinary protein loss decreases indicates clearly that proteinuria is most important in the pathogenesis of hypoproteinemia. However, there is reason to believe that other factors are concerned in this phenomenon. On occasion the daily protein loss may exceed 30 gm., but usually it lies between 5 and 10 gm.[74, 75] This appears to be relatively small in terms of protein replacement capacity. Co Tui[76] estimates, on the basis of changes observed in animals after plasmapheresis and in human blood donors after repeated blood letting, that normal man should be capable of manufacturing approximately 55 gm. of protein per day, provided an adequate diet is given. Some patients with the nephrotic syndrome can maintain a constant (though low) plasma protein concentration by manufacturing at least 20 gm. of protein per day to replace that lost in the urine. But others fail to replace even this much, and it may be posited that there is a fundamental impairment of protein synthesis in the nephrotic syndrome that varies quantitatively from case to case.

The evidence regarding this point is confusing, fragmentary and often contradictory. Very few patients can be or have been subjected to the necessary expensive and protracted balance studies. It is very difficult to be sure that the data of different studies are comparable. But it now seems certain that increasing the dietary protein intake does not usually result in augmented plasma protein synthesis. Berglund, Scriver and Medes[77] and others[78] have found that the plasma proteins increase slightly in response to high-protein feeding and that proteinuria may be remarkably exaggerated. Farr[79, 80] and others,[81] on the other hand, failed to find any change.

Similarly, disagreement is evident regarding basic nitrogen needs. In children it appears to be very difficult to maintain a positive nitrogen balance.[79, 80] The optimal intake is not much in excess of the needs of a child of the same age and ideal weight, and only slightly greater protein intake may have a toxic effect by reducing protein assimilation.[82] Administration of amino acids, by mouth or, better still, by vein, seems to counteract this effect to some extent.[83] In most adults nitrogen equilibrium is more easily achieved, although utilization of protein is determined by the caloric intake and by the character of the protein and amino acid mixture ingested. The divergencies between nitrogen needs for children and adults may be in part attributable to the factor of growth and, when this is of no importance, to differences in the extent of protein depletion.

The plasma proteins are believed by many workers to be in equilibrium with an extensive "protein pool" the bulk of which is extravascular.[84] Reduction of the plasma protein accordingly denotes reduction of the total labile body protein, and repair of a plasma protein deficit entails replacement throughout the pool as a whole. Very little is known of the measures employed by the body to protect the composition and size of the pool, though it appears that a fall in plasma protein stimulates manufacture and possibly reduces catabolism. Moreover, catabolism is lowered during starvation to a minimum commensurate with intracellular activity and caloric needs.[85] When an adequate caloric intake is assured and only nitrogen withheld, a still greater reduction occurs, though it appears that some 20 gm. of protein is destroyed even then each day. It is not known how protein degradation and nitrogen excretion are reduced in starvation, but the same processes are probably operative in the nephrotic patient, since, almost without exception, such patients lose tissue protein and present striking emaciation after removal of edema fluid therapeutically or by spontaneous remission. In experimental animals plasmapheresis has also been found to draw upon proteins essential in hemoglobin production.[86] Perhaps this explains the anemia seen in some cases of nephrosis. Hence, it seems reasonable to regard the nephrotic syndrome as a state of quasistarvation in which nitrogen requirement is reduced to a minimum. Nonetheless, the nephrotic patient has a nitrogen requirement considerably in excess of the sum of the theoretical minimum and the amount of nitrogen lost in the urine, possibly to meet the need of replenishing a depleted protein pool or to replace tissue protein.

It is not quite clear why the protein pool should be depleted. Certainly, many patients continue to have excellent appetites and to consume consider-

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‡Assistant resident, Presbyterian Hospital.

able amounts of protein food throughout the course of their illness. It must be assumed that very large quantities of protein are lost in the urine when the plasma protein level is still normal, before symptomatology demands the attention of the physician. Patients may be expected to differ markedly in the extent to which this process has been carried. Thus, in Berglund's[77] series a lag period of several days following the administration of a high-protein diet intervened before increased plasma protein synthesis became evident in a slight increase in plasma protein concentration and proteinuria. This period may represent the time required for the restocking of the extravascular component of the protein pool. In contrast, in only 1 patient studied by Keutman and Bassett[81] did the plasma protein level rise and then only because proteinuria slacked off. Hence, depletion may have been more marked in their patients. It is possible, of course, that these observations indicate the inadequacy of the protein pool concept in this connection, since nitrogen may be required for the building of stable proteins as well as labile components of the pool.

Theoretically, the ideal replacement material should be concentrated purified fractions of the human plasma proteins, but unfortunately these substances have often failed to increase protein concentration satisfactorily.[37, 38] Nor is there any evidence that protein synthesis is stimulated by the additional protein intake. One difficulty arises from the fact that the injected protein is immediately distributed throughout the protein pool. Luetscher[87] has found that a small percentage of injected albumin appears in the urine, that most of the remainder disappears from the blood within forty hours and that only a small fraction remains in the circulation. When one considers the probable size of the protein pool this behavior is not unexpected. In the starved dog the ratio between extravascular and intravascular protein is 25:1, indicating that a total increment of 25 gm. of protein is required to increase plasma proteins by only 1 gm.[88] If this ratio were 10 in nephrotic patients, it is evident that 300 gm. of plasma protein must be given to increase the plasma protein concentration by 1 gm. per 100 cc. Even when adequate protein is furnished, however, repletion appears impossible in many patients. Moreover, it cannot be explained why endogenous protein synthesis should not be sufficiently effective to make more than 5 to 10 gm. of plasma protein daily in excess of metabolic needs when adequate dietary protein and a high intravenous protein intake are assured.

More direct evidence that a disturbance of nitrogen metabolism occurs is found in the behavior of the amino acid level in certain cases. Farr and MacFadyen[89] have observed a chronic reduction in the plasma amino acid concentration (ninhydrin method), which is accentuated prior to and during the "nephrotic crisis" in children with the nephrotic syndrome. These episodes are characterized clinically by fever and abdominal pain occurring in association with a peritonitis for which, in many cases, there is no demonstrable infectious cause. Administration of amino acids parenterally is believed to have reduced the mortality of crisis.[90] Farr[89] thinks that the low level of amino acids may betoken "toxic destruction" of proteins since it is associated with a negative nitrogen balance and excessive nitrogen urinary loss. A similar hypoaminoacidemia has been observed in normal persons[91] during various infectious diseases and after trauma or operation. It is interesting that in these situations it is also attended by nitrogen loss and evidence of "toxic" proteinolysis. These phenomena cannot be explained at present, but it is possible that the patient with the nephrotic syndrome suffers continuously a similar defect in nitrogen metabolism that interferes with protein production.

It is of interest that dietary protein restriction coupled with plasmapheresis in dogs results in a marked hypoproteinemia characterized like the nephrotic syndrome by a marked decrease in the albumin fraction.[92] The maintenance of the plasma globulin concentration despite equivalent loss probably indicates more efficient replacement. It seems not unlikely that the failure of plasma globulin to fall in nephrosis in the presence of a depleted protein pool may likewise imply continued normal globulin synthesis and whatever defect there may be must concern albumin synthesis directly.

The principal site of albumin manufacture is believed to be the liver.[84] Few studies of liver function have been made during the nephrotic syndrome. Lyttle et al.[93] found that nephrotic patients removed injected amino acids from the blood as readily as normal persons, whereas children with liver disease failed to do so. Moreover, Kirk[94] has shown that deamination of glycine proceeds normally in this disease. Galactose and bromsulfalein removal have been observed to be normal or occasionally only slightly impaired in other patients.[95] It is possible that these discrete activities are undisturbed even though hepatic protein synthesis is seriously deranged.

Since nephrotic patients appear to secrete little sulfur relative to nitrogen, it has been suggested that "deposit" protein[96] — characterized by a low sulfur content, presumably in the intracellular compartment of the protein pool — is chiefly concerned in urinary protein loss. Grabfield[96] considers a defect in the intermediary sulfur metabolism to be essential to the pathogenesis of the nephrotic syndrome. However, his work is based upon the total nitrogen and sulfur content of the urine. Hence it is possible that excessive excretion of nonprotein nitrogen is the cause for the observed reduction in nitrogen-sulfur ratio rather than a truly low nitrogen-sulfur ratio of urinary protein. Studies of nitrogen and sulfur metabolism with radioactive tracers

in the nephrotic syndrome are awaited with great interest.

Epstein[97] has called attention to the possibility that a disorder of thyroid activity is involved, since the basal metabolic rate is frequently reduced. It is now generally believed, however, that this finding is of questionable significance. Edema[98] and the state of quasistarvation[85] may suffice to reduce the basal metabolism. Treatment with thyroid extract and thyroxin has proved disappointing.[14, 85, 99]

Insufficient evidence is at hand to prove conclusively that a faulty protein metabolism, particularly in plasma albumin synthesis, is in part responsible for hypoproteinemia. The irregularity of protein production and excretion in different patients, the failure to replace protein despite adequate and proper nitrogen supply and the signs of "toxic" protein destruction, including hypoaminoacidemia and negative nitrogen balance, seem to indicate this possibility. Nonetheless, protein loss in the urine is probably of the greatest importance since it appears to be essential to the development and maintenance of a low concentration of protein in the blood.

Edema

From the standpoint of the patient, edema is the most distressing and disabling manifestation of the nephrotic syndrome. The distribution of subcutaneous fluid appears to be governed by gravity and tissue tension. Burch[100, 101] has shown that the predilection for the eyelids (particularly the lower lid) may be attributed to the low tissue pressure and the high distensibility of the skin in this area. Failure of lymphatic drainage when the eyelids are motionless and not blinking, as during sleep and during recumbency, explain the tendency for edema fluid to accumulate about the eyelids during the night. The genitalia and loose abdominal wall of many multiparas present similar characteristics and are often excessively edematous. This fluid may be under considerable pressure since the skin over the lower extremities may become shiny white and quite taut, often cracking and splitting under the strain. Edema fluid drains profusely through such breaks in the skin, and secondary infection, especially dangerous in these patients, may follow. Great discomfort may result from the restriction of movement. Pleural effusion or ascites often reduces the vital capacity greatly, and acute pulmonary edema occasionally appears. Localized edema of the glottis may prove rapidly fatal. Edema of the gastrointestinal tract[102] may account for radiologic evidence of delayed gastric emptying time and decreased motility of the small intestine.

The pathogenesis of nephrotic edema is not yet settled. Many discussions of this question fail to consider it in its entirety. Importance is attached, on the one hand, to the plasma protein concentration in relation to the osmotic pressure acting across capillary membranes throughout the body as a determinant of the volume of interstitial fluid and, on the other, to renal activity in excreting or retaining water and electrolytes. Obviously, both factors are interrelated and interdependent. Neither should be stressed unduly to the exclusion of the other.

There is little doubt that a lowered plasma protein concentration results in the formation of edema.[17, 103, 104] Leiter[105] and others[106, 107] have proved conclusively that the removal of plasma proteins and low-protein diets lead to persistent hypoproteinemia and the development of edema in experimental animals. Starling's[108] hypothesis that the distribution of extracellular fluid is dictated by balanced transcapillary pressures — plasma oncotic pressure and tissue tension opposed to interstitial fluid oncotic pressure and capillary blood pressure — has been applied in explaining this phenomenon, in particular by Epstein,[6] who is largely responsible for the following viewpoint. Other things being equal, a reduction in plasma protein concentration connotes reduction in plasma oncotic pressure. This is even more likely in the nephrotic syndrome, in which the proteins of greatest osmotic activity are specifically withdrawn from the blood.[109] Moreover, many measurements of plasma osmotic pressure[110–112] reveal far greater diminution in the nephrotic patient than can be accounted for on the basis of the technical errors of the relatively crude methods thus far employed. Hence it may be presumed that the fall in oncotic pressure leads to increased filtration of water and diffusible solutes into the interstitial spaces, with an expansion of extracellular extravascular water volume and the formation of edema. The factors concerned in effecting a new equilibrium of the transcapillary forces are not easily discerned. Possibly, filtration in excess of reabsorption continues until an increment in tissue tension corrects the deficit in oncotic pressure.

According to this hypothesis the movement of fluid into the tissue spaces must occur at the expense of the plasma volume. The published figures for plasma volumes are conflicting.[113–117] In part, this may be attributed to errors introduced by lipidemia in various dye-dilution technics.[37] And in part it appears to be a result of a true variability in plasma volume. In accord with theory, plasma volume appears to be reduced in most cases. The most reliable studies, based upon measurements with carbon monoxide, indicate that the reduction of plasma volume is much less when anemia is a complicating factor. With diuresis, the plasma volume rises above normal in such cases, accounting, perhaps, for divergent observations.

Epstein's hypothesis is weakened considerably by observations that diuresis and loss of edema may occur in the absence of any detectable change in plasma protein concentration.[118] Plasma osmotic pressure may even fall. Similarly, in hunger edema, which resembles nephrotic edema in many respects,

the extracellular water volume may expand or contract without reference to protein concentration,[119] though it is claimed that osmotic-pressure changes are of chief importance.[120] Here, again, attention has been focused upon possible changes in the peripheral circulation. Keys[119] suggests that the capillary wall is in a state of "dynamic non-equilibrium," an assertion that is somewhat difficult to understand. Increased capillary permeability would increase effective filtration pressure across capillary walls and lead to edema formation. However, evidence that this occurs is unsatisfactory. The edema fluid and transudates contain very little protein, usually less than 0.5 per cent.[121] On the other hand, Luetscher[87] has found that injected albumin rapidly disappears from the blood, and Lange and his co-workers[122] claim that fluorescein bound to protein passes easily into the edema fluid. These observations require critical testing before they can be accurately evaluated. A third of the osmotic activity of plasma proteins depends upon their electrical charge and the resulting unequal distribution of diffusible ions on each side of the capillary membrane (the Donnan equilibrium).[109] A change in this characteristic, thus far not detected, may be a factor in determining equilibrium conditions in the capillaries and the distribution of extracellular water. Perhaps alterations in the capillary wall are influential in affecting this factor. Changes in over-all tissue tension and capillary pressure cannot be appraised with accuracy by the methods at present available.

Moreover, the use of albumin concentrates to repair the deficit in plasma albumin has proved rather disappointing.[37, 38, 123] Diuresis and mobilization of edema may occur in some cases, but the effect is often limited and unimportant. Marked increases in plasma volume may prove detrimental and prevent theoretically appropriate therapy, a development that would not be expected if reduced protein concentration were the sole important defect.

Opposed to the view that changes in the blood or at the periphery have primacy in determining the volume of extravascular fluid is the belief that renal dysfunction in the disposal of water and electrolytes is chiefly concerned. Certainly retention of water by the kidneys is required and does occur. One school of thought posits a specific fault in renal excretion of sodium.[124, 125] This claim is now supported by a considerable body of evidence, but it has been clearly proved that the nephrotic patient handles sodium in a manner that differs quantitatively rather than qualitatively from the normal.[118] Indeed, the normal kidney, in the face of a water and sodium deficit, acts like the nephrotic kidney. Hence the nephrotic patient appears to be in a state of quasidehydration, which might be explained by diversion of water and electrolytes from the plasma into the interstitium. In addition, there are disturbances of renal function that may contribute positively. The glomerulotubular imbalance, described above, might be expected to predispose to retention by enhancing water and salt reabsorption in excess of the body need. Diuresis, either spontaneous or induced by intravenous injection of salt-poor human plasma albumin concentrates, has been observed to occur in association with an increase in glomerular filtration rate.[38, 87, 126] The resulting reversal of the imbalance should be important in the explanation of the increased output of urine. The possibility that increased formation of antidiuretic hormone by the posterior pituitary is responsible in part for the water retention has been suggested by the appearance of antidiuretic substances in the urine.[127] There is no evidence that the increased tubular reabsorption of sodium is a result of adrenal cortical dysfunction.

The alleviation of the nephrotic syndrome during various infectious diseases has frequently been observed.[128–130] Clément[128] has described regression of edema in response to the pyrogenic reaction following administration of typhoid vaccine, and deliberate infection with measles has been suggested as one means of treating the disorder.[129] Unfortunately, the effect of fever cannot always be predicted. In many patients, it proves definitely detrimental, inducing hematuria and further accumulation of edema. Moreover, patients with the nephrotic syndrome are peculiarly lacking in resistance, usually dying as the result of some intercurrent infection. Hence, the pyrogenic reaction must be condemned as a therapy, but it is of great interest in connection with the problem of the pathogenesis of nephrotic edema. Fever gives rise to striking changes in the renal and peripheral circulations. The cardiac output rises, and blood flow through the kidney and skin increases, apparently as a result of vasodilation. How these changes are related to the withdrawal of tissue fluid and diuresis is unknown, but they indicate that hemodynamic factors play an important role.

It is evident that knowledge of nephrotic edema is fragmentary. Many unassailable facts are at hand, but they cannot be fitted as yet into any satisfactory theory. Probably several variables are involved. The accumulation of fluid in the tissue spaces undoubtedly denotes an imbalance of transcapillary pressures and simultaneous water and electrolyte retention by the kidney. Mobilization of edema requires an adjustment in the periphery to promote the flow of fluid into the blood and a change in renal function to permit its excretion. Unilateral activity thus appears unlikely, but the fundamental activities in the peripheral vascular bed and the kidney are obscure.

Lipidemia

One of the most puzzling manifestations of the nephrotic syndrome is the disturbance of fat

metabolism that gives rise to hypercholesterolemia and hyperlipemia. The elevated concentration of fatty materials may render the plasma opaque and milky in appearance, and greasy in consistence. There is a tendency for the fat to separate slightly on standing, and part of it may be removed by centrifugation, but a considerable quantity appears to be loosely attached to protein or to serve as a coating of protein aggregates,[63, 131] so that complete physical separation may be impossible by ordinary means. Cholesterol is a relatively insoluble substance, and yet 1 or 2 gm. may be carried in solution in each 100 mm. of nephrotic plasma. Such quantities cannot be dissolved in normal plasma, but even larger amounts can be held in solution in the nephrotic plasma.[132]

No clinical abnormality can be definitely ascribed to lipidemia. Certainly, atherosclerosis occurs more frequently among nephrotic patients than in normal persons of the same age.[133] It is in children with this disorder that one sees the most advanced forms of childhood arteriosclerosis. There appears to be a relatively constant coincidental, but by no means obligatory, relation to hypoproteinemia and edema. When edema clears, the plasma lipids tend to return to normal levels, and if edema continues to be evident, lipidemia may persist even though uremia develops.[134]

Plasma cholesterol is always elevated. Levels as high as 2.0 and 2.3 gm. per 100 cc. have been observed. All the other lipid fractions are likewise increased. Cholesterol esters appear to rise in most patients to the same extent as free cholesterol.[85] Peters and Man[135] found that the phospholipids (lecithin, the cephalins and other phosphorus-containing lipids) parallel cholesterol, whereas Thomas[136] observed a somewhat closer relation between lipid phosphorus and the total lipid content of the plasma. The neutral fats are always increased, and it has been observed that they tend to make up a larger fraction (as much as half) of the total lipids as the disease advances.

The fat content of the diet appears to have little influence upon the plasma composition. Hiller et al.[137] and others[138] observed slightly greater increments in the plasma fatty acid and lecithin content after fat ingestion than in normal subjects or nephritic patients, but it appears that nephrotic patients can burn fats as efficiently as normal persons, since the respiratory quotient decreases in a normal manner when fat makes up the greater portion of the caloric intake.

The causes of lipidemia in nephrosis are quite obscure. In large part this is a result of the confused state of knowledge regarding normal fat metabolism. Since nephrotic patients appear to absorb and utilize dietary fat in a normal manner it has been suggested that lipidemia represents a failure of the processes of fat removal from the blood into storage sites.[137] Contrariwise, Thannhauser[139] claims that lipidemia is due to enhanced mobilization of fat from the body stores. There is no evidence in support of either hypothesis. Since wasting of the tissues is prominent, lipidemia has been likened to that occurring in starvation, but in chronic malnutrition the cholesterol content of the plasma is usually depressed.[85] The possibility that a disorder of the thyroid gland is involved has likewise been considered[140] and found wanting, since there is no response to specific therapy and reductions in the basal metabolic rate may be apparent rather than real owing to a failure to use "ideal" body weights in calculations.[141] Heymann and Clark[142] and Winkler and his co-workers[143] have induced lipidemia in animals by bilateral nephrectomy and ligation of the ureters. Lipidemia also develops after nephrotoxic nephritis[144] and subtotal nephrectomy.[145] The cause of this phenomenon is unknown, but it does not seem to have any relation to the problem in nephrosis, in which renal function is usually relatively unimpaired. It appears that lipidemia must be considered a phenomenon connected in some way as yet unknown with the hypoproteinemia and edema of the nephrotic syndrome. Little light has been thrown upon this question by the experimental production of hypoproteinemia in animals. In this situation the development of lipidemia is apparently attributable to hepatic insufficiency,[146] but in the nephrotic syndrome, hepatic function appears to be normal.

* * *

As a rule the nephrotic syndrome is distinctive, presenting each of the specific disorders discussed at length above. It should be emphasized, however, that it may be complicated by other independent disturbances, such as congestive heart failure, uremia and hypertensive heart disease. Extraneous manifestations that mask or distort the usual clinical and physiologic pattern may thus be introduced. Finally, the syndrome itself presents a shifting and variable course. The most striking clinical phenomenon, edema, may be minimal and even absent from time to time. Proteinuria, hypoproteinemia and lipidemia are fundamental derangements, and so long as they persist the nephrotic syndrome may be considered to be present. This concept is important in the interpretation of therapeutic effects since transient elimination of edema cannot be considered as evidence for the curative power of any agent.

Another confusing aspect of the nephrotic syndrome is the diversity of its causes. The typical complex of symptoms and signs may develop during syphilis and clear completely in response to antibiotic or arsenical therapy. Chronic diffuse glomerulonephritis, intercapillary glomerulosclerosis, amyloid disease and other disorders give rise to the same clinical picture. Each of these may be distinguished only by the characteristic renal lesion

or clinical course, or both. In only one disorder, lipoid, "genuine" or chronic nephrosis, is there no anatomic change in the kidney. Since even in this condition proteinuria and other manifestations appear to depend upon an abnormality of glomerular membranes, lipoid nephrosis may also be primarily a renal disease.

In any case, the nephrotic syndrome can be profitably viewed as a discrete entity. As such it remains an unsolved riddle. Secondary infection, which was formerly devastating in these patients, can now be combated with powerful chemotherapeutic agents, but specific therapy is altogether lacking. It can only be hoped that the use of newer methods of study in the clarification of the metabolic, hemodynamic and renal-function disturbances, of which the available evidence is indicative rather than demonstrable, may yield a clue to the means of effective treatment.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

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CASE 34081

PRESENTATION OF CASE

A forty-nine-year-old man entered the hospital because of difficulty in urination.

For two months he had frequency (ten times during the day and five times at night). This was associated with weakness and hesitancy of the stream. There was no dysuria, gross hematuria, back pain, feverishness or vomiting. One week before entry he had an attack of dyspnea, wheezing and cough, and he developed conjunctival and palpebral hemorrhages. The blood pressure was 120 systolic, 80 diastolic. The legs became edematous.

Physical examination revealed a pale drowsy man. The face was puffy, and there were conjunctival and palpebral hemorrhages. The eye grounds revealed flame-shaped hemorrhages, narrow arteries and blurring of the disk margins. The neck veins were distended and pulsating. There was an apical systolic murmur. In the abdomen there was a fluid-filled, tense, nontender mass extending upward from the pelvis to the level of the umbilicus. Rectal examination revealed tender external hemorrhoids and a questionably enlarged prostate. The legs and feet were edematous.

The temperature was 99°F., the pulse 88, and the respirations 16. The blood pressure was 175 systolic, 90 diastolic.

Examination of the blood disclosed a hemoglobin of 5 gm. and a white-cell count of 7300. The nonprotein nitrogen was 200 mg., the calcium 6.8 mg. and the phosphorus 8.2 mg. per 100 cc.; the alkaline phosphatase was 3.0 units, and the carbon dioxide was 10.6 milliequiv. per liter. The prothrombin time was 18 seconds (control, 17 seconds). Repeated examinations of the urine gave a reaction of pH 6.0 to 7.0, with a specific gravity of 1.008 to 1.014, and a + to ++++ test for albumin. The sediment showed many red cells and on one occasion 20 to 30 white cells per high-power field. Cultures grew colonies of *Staphylococcus aureus* and colon bacilli.

An electrocardiogram showed digitalis effect only. An x-ray film of the chest revealed basal congestion of the lungs and some left ventricular hypertrophy. A plain film of the abdomen was normal.

The patient was catheterized in the Emergency Ward, and 750 cc. of clear urine was removed. A few hours later, after several unsuccessful attempts to pass a No. 18 catheter, a No. 20 Foley catheter was passed without difficulty, and 1000 cc. of clear urine removed. There was a good deal of subsequent bleeding into the bladder, and clots were removed by irrigations as required. He was digitalized and given resuspended red cells in 10 per cent dextrose in water. Fluids were forced to 4000 cc. a day. The palpebral hemorrhage and edema gradually subsided. After a few days the indwelling catheter became very painful. He began to vomit, and fluids were then pushed intravenously and by proctoclysis. The uremia and acidosis increased. Repeated nonprotein nitrogen estimations were 175, 250, 210 and 280 mg. per 100 cc., with corresponding carbon dioxide estimations of 10.6, 14.7, 13.4 and 17.5 milliequiv. per liter. He died on the tenth hospital day.

DIFFERENTIAL DIAGNOSIS

DR. FLETCHER H. COLBY: May we see the x-ray films?

DR. STANLEY M. WYMAN: The heart shows enlargement, chiefly of the left ventricle. The aorta is unusually tortuous for a man of this age. The appearance suggests hypertension. There is an

area of ill defined, hazy density in the middle thirds of both lung fields, with slight accentuation of the basilar markings. No unusual soft-tissue masses are visible in the film of the abdomen, and there are no unusual areas of calcification.

DR. COLBY: Can you make out the renal outlines at all?

DR. WYMAN: I cannot make them out with certainty.

DR. COLBY: You cannot say whether or not they are enlarged?

DR. WYMAN: Not with enough certainty to rely on. My guess is that they may be slightly small.

DR. COLBY: The fact that a large catheter passed more easily than a small one is of no great significance — it is not unusual for a patient to have enough spasm of the sphincter muscles for that to happen. The subsequent bleeding into the bladder is not unusual. Any overdistended bladder may bleed in this manner when rapidly emptied.

When the patient entered the hospital he obviously was critically ill. All the signs and symptoms that we are given point to a lesion of the heart, as well as to some degree of urethral obstruction, and certainly to severe renal damage. All these signs and symptoms may be integrated, although I cannot be certain.

Let us take up the obstructive feature first. The first possibility is some sort of congenital abnormality, which should have made itself evident before the age of forty-nine, however. Such lesions as congenital valves and congenital stricture of the urethra occur in young persons and become evident before this age. A stricture of the urethra can be eliminated by the fact that the patient had nothing in the past history to suggest it, and in the Emergency Ward he was easily catheterized. A catheter cannot be passed easily by a stricture of any severity.

Does a patient of forty-nine have benign hypertrophy of the prostate? He may. I recently operated on a man of forty-nine who had a definitely obstructed prostate with a stone in the bladder. I said, "You are young to have this." And he answered, "I know it; my brother is fifty, and last year he had his prostate operated on."

This man entered the hospital because of difficulty in urinating and with a past history of weakness and hesitancy of the stream. Certainly, prostatic hypertrophy is a definite possibility, and probably infection as well. The dyspnea, wheezing and cough, if not asthmatic (and apparently they were not), suggest cardiac disease. This is confirmed by the systolic murmur, distended neck veins, pulmonary congestion and left ventricular hypertrophy. We can therefore say that he had hypertensive heart disease. One thing that bothers me a good deal is the fact that the blood pressure was 120 systolic, 80 diastolic, and a week later 175 systolic, 90 diastolic. Is that correct?

DR. EDWARD F. BLAND: Not quite. On admission it was 200 systolic, 100 diastolic.

DR. COLBY: There was not the change from 120 to 175 systolic in a week?

DR. BLAND: A later reading was 165 systolic, 90 diastolic, and as the patient became sicker the pressure was lower.

DR. FREDERICK C. GOETZ: The blood pressure was 125 systolic, 70 diastolic, about a week before he entered the hospital, according to his doctor.

DR. COLBY: Was he pretty sick then?

DR. GOETZ: Yes; he was.

DR. COLBY: The general signs and symptoms are those of a marked degree of renal damage in a patient who was not able to concentrate urine above 1.014. He had a low specific gravity of urine with albumin, a low serum calcium, a high phosphorus and marked anemia. The marked anemia must mean a disease of long standing. The hemoglobin was 5 gm. He also had eye signs and edema. It seems unlikely to me that such a degree of renal change in a man of forty-nine could have been associated with a hypertrophied prostate, so that I think we have to look for the chief lesion in the kidneys.

What sort of lesion could it have been? The lack of pain in the past and the other symptoms seem to eliminate a certain number of renal lesions, such as hydronephrosis and calculus disease, and the obstructive lesions that affect the upper urinary tract. On the other hand this must have been a bilateral affair, and it seems to me that the most likely possibility is a severe degree of chronic pyelonephritis. This is borne out by the urinary findings, the low specific gravity, the edema, the obviously failing renal function, the eventual acidosis and death and uremia. He had a mixed infection, staphylococcus and colon bacilli in the urine, which is quite consistent with marked pyelonephritis. Acute pyelonephritis is much more apt to have only one organism.

One condition that I cannot rule out, although I have no evidence to favor it except a pair of kidneys that were failing, is bilateral polycystic disease, which is always a possibility. This man was at the age when polycystic disease is apt to make itself evident. There was nothing in the past history to suggest it, however, such as back pain, gradual rise in blood pressure and attacks of gross hematuria. But I cannot eliminate the possibility. That is why I asked particularly about the x-ray findings, to see if we could get any additional information from Dr. Wyman.

My diagnoses are therefore hypertensive heart disease, benign hypertrophy of the prostate and uremia due to pyelonephritis. The patient probably had a pair of lungs that showed quite a bit of edema.

DR. TRACY B. MALLORY: Can you add anything, Dr. Bland?

DR. BLAND: I cannot add anything diagnostically to what Dr. Colby has said. However, the service deliberated at length regarding treatment. Here was a man with uremia, swollen neck veins and extensive edema. The one finding that I would like to emphasize is the low hemoglobin, and we have only recently appreciated that under this circumstance one can get cardiac failure with a high cardiac output and swollen veins. Hence, whatever we could do to improve the hemoglobin level might help him. In spite of the congestive failure, we gave him washed red cells, and he was on a rigid low-sodium regime. The hemoglobin increased from 5 up to 10 gm. The distention of the veins actually lessened as the hemoglobin improved. The edema was not particularly influenced, and he slowly worsened in the course of ten days and died in uremia. We did not know the exact nature of the difficulty in the genitourinary tract, because he was so desperately ill throughout the stay on the ward that further study was inadvisable. We thought it was due to obstruction, back pressure on the kidneys and, most likely, infection.

CLINICAL DIAGNOSIS

Uremia, cause unknown.

DR. COLBY'S DIAGNOSES

Hypertensive heart disease.
Benign hypertrophy of the prostate.
Uremia, due to pyelonephritis.

ANATOMICAL DIAGNOSES

Benign prostatic hypertrophy, with obstruction.
Hypertrophy of bladder.
Hydronephrosis, bilateral.
Parathyroid hyperplasia, ascending, slight.

PATHOLOGICAL DISCUSSION

DR. MALLORY: This man died purely and simply as the result of prostatic obstruction, despite his comparative youth. The story reminded me of a graduate student I had a number of years ago who shortly after he arrived impressed me as being far less bright than the average. Then I began to fancy that I smelled a slight uremic breath and referred him to Dr. Chute, who found that he had an enlarged prostate, with severe obstruction, at the age of forty-three years. Following a transurethral resection his mentality improved considerably.

The prostate in the case under discussion was symmetrically enlarged, and the bladder was enormously hypertrophied, the muscular wall measuring over 1 cm. in thickness. There were numerous incipient diverticula but none that penetrated through the entire thickness of the bladder wall. Each ureter measured 1.5 cm. in circumference. The kidneys were small. The pelves were grossly dilated, but the cortex of each kidney ranged from 0 to at most 3 mm. in thickness. All traces of pyramids had disappeared.

Microscopical sections of the kidney showed almost complete atrophy of the tubules, persistent glomeruli and minimal interstitial inflammation — a characteristic picture of hydronephrotic atrophy. The lungs at autopsy weighed 800 gm., which indicates about as little pulmonary edema as one can hope for in a patient dying in uremia. The heart was within normal limits, and we found nothing wrong with it.

CASE 34082

PRESENTATION OF CASE

A sixty-four-year-old housewife entered the hospital because of anorexia.

Ten months previously, following an emotional upset, precipitated by the mysterious disappearance of her daughter, she experienced an episode of depression, associated with anorexia, with rather slow recovery. Shortly after this, an attack of "biliousness," with nausea, vomiting and mild epigastric pain ensued, with mild icterus and pruritus, but without chills, fever, dark urine or light stools being recognized. Three months before entry she had experienced headache and weakness and following treatment for anemia with ferrous sulfate, thiamine and liver injections she improved markedly in strength and appetite. Two gastrointestinal series during a six-week interval prior to admission, however, had demonstrated a constant filling defect, with ulceration, in the prepyloric region.

Physical examination disclosed an old left simple mastectomy scar. There were bilateral varicosities, with slight edema of both lower legs. The examination was otherwise negative.

The blood pressure was 220 systolic, 115 diastolic.

Examination of the blood revealed 13.5 gm. of hemoglobin and a white-cell count of 8200, with 80 per cent neutrophils. Examination of the urine was negative. An x-ray film of the chest was normal. A gastrointestinal series revealed a definite abnormality involving the antral portion of the stomach, with a filling defect of rather smooth contour measuring almost 3 cm. in diameter involving the posterior wall. A small ulcer crater was observed toward the proximal portion of the defect. There were fairly good peristaltic waves in this region. A gastric analysis was negative for free hydrochloric acid on a fasting specimen and one hour following histamine injection.

On gastroscopic examination normal peristaltic waves were seen passing down over a normal-appearing antrum, until they reached a submucosal lesion on the lesser curvature close to the pylorus. This lesion appeared to be about 2.5 to 3 cm. in diameter,

with a definitely smooth, normal-appearing mucosa over it, elevated above the surrounding mucosa by about 1 to 1.5 cm. There was very little distortion of the peristaltic wave. No ulcer was visible on its surface.

An operation was performed on the ninth hospital day.

Differential Diagnosis

Dr. Claude E. Welch: This is the record of a patient with a gastric lesion of an undetermined type with incidental hypertension, a mastectomy scar and varicose veins. The history of jaundice is very equivocal, and no further signs or symptoms later developed to implicate the biliary tract. The only other abnormal feature in the history or physical examination is that of slight edema of both lower legs. Since this patient was subjected to no further examination to clarify the cause of the edema, with the exception of one negative examination of the urine, I believe that we can dismiss this symptom as inconsequential.

We have, then, a history of anorexia that began ten months before admission. This was more or less persistent and was followed by epigastric pain at an indefinite date. The history is also very suggestive of an anemia due to blood loss that developed three months before entry.

The diagnosis of prepyloric ulceration was first made about six weeks prior to entry. It may be assumed that following this discovery the patient was treated for a peptic ulcer. However, the filling defect persisted, and was observed at a second examination six weeks later. This filling defect was again observed when the patient entered the hospital.

The presence of achlorhydria that was persistent despite the injection of histamine is of great importance, since nearly 50 per cent of the patients with this finding and a gastric ulceration are found on pathological examination of the resected specimens to have cancer of the stomach.

The x-ray and gastroscopic findings were as usual somewhat at variance. Both of them suggest a submucosal lesion on the lesser curvature in the antral area. However, the presence of ulceration that was noted three times on the x-ray examination was not observed by the gastroscope. Under these circumstances and in this location, I favor the x-ray description and consider it definite that this patient had an ulcerating lesion. That it was associated with a submucosal mass is quite likely, but not nearly so important as the presence of the ulceration itself.

There are only a few diagnoses to be considered. The ulcer must have been a benign gastric ulcer, carcinoma or the tiny ulceration that is frequently found over pancreatic rests or spindle-cell tumors. The evidence of a submucosal mass could point to a spindle-cell tumor, a pancreatic rest or a tumor that is projecting into the wall of the stomach from the lesser peritoneal cavity. In this location, of course, the most likely source is the pancreas. It will be noted that the ulceration appeared on x-ray study prior to the time that the larger filling defect first appeared. It could therefore have represented an extension of carcinoma into the submucosal area or even outside the stomach.

The salient features of a prepyloric lesion of short duration developing in a patient in the older age group, the ulceration being associated with complete achlorhydria and failing to heal under at least six weeks of medical treatment, all point to the diagnosis of carcinoma of the stomach rather than to benign ulcer or the rarer tumors mentioned above.

Dr. Milford D. Schulz: All films made of the stomach show a sharply delimited defect on the lesser-curvature side of the antrum, measuring only a few centimeters across. The mucosal pattern stops abruptly at the defect, which looks as though it is caused by a smooth mass projecting into the stomach lumen. I am not certain about the ulceration described — it may be just barium caught in a fold. If there is ulceration, it must be in a tumor mass, but whether the tumor is intraluminal or intramural is not certain, though from the films I should suspect the tumor to be intraluminal.

Incidentally, there is a small diverticulum of the duodenum lying just next to the antral defect.

Dr. Edward B. Benedict: It interests me that Dr. Welch should say that the x-ray and gastroscopic findings were "as usual" somewhat at variance. As a matter of fact, in the easy cases they usually agree; in the difficult ones they may not. In a review of 298 cases of gastric carcinoma, gastric ulcer, jejunal ulcer, gastritis, benign tumor, lymphoma, sarcoma, metastatic carcinoma and normal stomach studied by x-ray and gastroscopy and proved by pathological examination, it was demonstrated that the radiologist and gastroscopist agreed in more than half the cases.[1,2] It was also shown that, provided the gastroscopist obtains a good view of the lesion, his chances of reaching a correct diagnosis are greater than those of the radiologist. In this case the view was good, and there was no doubt in my mind that the lesion was submucosal. In my experience, nonulcerating submucosal tumors of the stomach have always been benign, with the exception of 1 case of carcinoma of the pancreas invading the stomach submucosally. Because of the vomiting, the proximity of the tumor to the pylorus and the possibility of cancer I felt that the lesion should be resected. If pathological study of the resected specimen demonstrates an ulcerating tumor, it shows how easy it is for the gastroscopist to miss an ulcer in or beyond a tumor. If, however, this proves to be a nonulcerating malignant submucosal tumor of the stomach, I believe it is exceptional. Whatever the pathological report, it is usually wise to

follow the rule that all tumors of the stomach are malignant until proved otherwise.

Clinical Diagnosis

Benign tumor of stomach, ? leiomyoma.

Dr. Welch's Diagnosis

Carcinoma of stomach.

Anatomical Diagnoses

Adenocarcinoma of stomach.
Pancreatic cyst.

Pathological Discussion

Dr. Benjamin Castleman: Will you tell us what was found at operation, Dr. Risley?

Dr. Thomas S. Risley: At operation the first abnormality encountered was an orange-sized cyst lying in the head of the pancreas just above and immediately adjacent to the lesser curvature of the stomach just proximal to the pylorus. The stomach was carefully palpated but no abnormality was felt. In the process of shelling out the cyst the lesser curvature was forced up farther, and it was only then that a freely movable mass about 1 by 1 by 3 cm. could be felt in the submucosa of the lesser curvature about 4 cm. above the pylorus. The mass could be moved over a distance of about 3 cm. beneath the muscularis. The stomach was therefore opened, and the mass that I felt externally appeared to be a thickened rugal fold, without any definite ulcer of the mucosa. This was excised for examination.

Dr. Castleman: We were called to the operating room to examine this specimen, which on gross examination was a section of the entire wall of the stomach with normal-appearing serosa and muscularis. The mucosa was a bit granular but could be moved over an ill defined submucosal thickening. No definite ulceration of the mucosa was seen. A rush frozen section revealed definite adenocarcinoma, and a subtotal gastrectomy was performed.

Our final examination showed that the entire lesion had been resected at the original procedure, because we could find only gastritis in the rest of the stomach. The regional lymph nodes were not involved. Permanent microscopical sections of the tumor showed a well differentiated adenocarcinoma involving the mucosa and part of the submucosa. The entire lesion was not more than 2 by 1 by 1 cm., and since there was no involvement of the muscularis or lymph nodes, one should expect a cure. It was one of the smallest gastric carcinomas that I have ever seen. The pancreatic cyst was thin walled, showed no papillary projections and was lined with epithelium similar to that seen in the pancreatic duct. Dr. Risley, do you believe that the cyst had anything to do with the symptoms?

Dr. Risley: The cyst was so near the stomach that it is conceivable that it could have caused pressure symptoms. However, the involved rugal fold was so near the pylorus that it probably caused the symptoms even though it was small.

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THE COST OF FELLOWSHIP

THE Council of the Massachusetts Medical Society on February 5, 1947, voted to increase the annual assessment of the fellows to twenty-five dollars, this increase to become effective on January 1, 1948. The vote was published in the *Journal* on April 17, 1947, and was discussed by the president of the Society in his address delivered at the annual meeting on May 19.

Five dollars of the increase of each fellow's assessment was allocated to the Boston Medical Library, and each fellow of the Society becomes thereby a Massachusetts Medical Society member of the Library. Dr. Phippen's letter of welcome is published herewith.

Other than this support of the Library, so necessary to that institution and so willingly given by the Society, the additional twenty dollars of the entire current assessment may require some accounting. We have but to look around us at the growing complexity of existence in an unsettled world to see the mounting costs of all of life's activities, and some of the reasons for them.

The increasing work of the Society is concerned mainly with education both of the profession and of the public, with constant regard to its public relations, and with protection of the health of the public and of the interests of the physician. Legislation must be vigilantly watched, and the ramifications of social medicine need unremitting attention.

Much of this work is done by the Society's numerous committees of unpaid and devoted members, but the work of these committees often entails considerable expense. Their activities have reached the point where they can no longer be co-ordinated by a part-time secretary. Our secretaryship must become a full-time position. Already the work of the committees and officers is showing the effect of the supplementary activities of our invaluable director of medical information and education.

The postgraduate lecture course in Cambridge, offered gratis by the Society, will shortly begin; the Bureau of Clinical Information finds a broadening field in discovering the right opening for the proper applicant; the Society has negotiated a pension plan for its employees of long standing; the office work increases week by week; fellows are offered legal defense against malpractice suits; each fellow in good standing receives, as an indissoluble part of his membership, a weekly journal for which the current annual subscription rate is seven dollars; the allotment to each district society has been increased.

The Society is growing up, and as it grows it finds more and more work to do in maintaining the standards, admittedly high, that have been established for the protection of both public and profession.

The increased assessment, still well below that of many state societies, is badly needed if the Society is to give proper service to its membership.

VULNERABLE GERM PLASM

IN a letter published elsewhere in this issue of the *Journal*, Dr. Abraham Myerson calls attention to a very important matter — the fact that radiant forces, which are now a leading factor in the advance

of biologic knowledge and medical treatment, have been clearly shown by Professor H. J. Muller,[1, 2] to produce deleterious hereditary mutations. The physician is interested in the welfare of the individual, but the scope of his activities and concerns must include the race if he is to avoid the reproach of the eugenicists that his efforts lead to the final injury of the race. In this, human beings are not exempt from the dangerous results of the advances of science in general.

What is of great importance both theoretically and practically is that a misleading dichotomy must now be discarded — namely, that hereditary and environmental agencies are separable in any complete sense. Germ plasm, the substances of ovum and sperm, is relatively stable, but that stability depends on a stable environment. The inner environment, such as the blood stream, is as necessary to the germ plasm as to the cells of other tissues and organs of the body. There is a vast experimental literature on this subject, including the influence of the vitamins, especially A and E, on the maturation and health of sperm and ovum, that is incontestable.[3-12]

Moreover, Mendelian heredity is fast becoming only a part of genetic knowledge. We are passing into a phase of great scientific advance, involving the physiology and biochemistry of the genes and of the cytoplasm of the gonads as well.[13] The nucleoproteins,[14] for example, are as basic in transmitting the qualities of hereditary nature as they are in the life history of the individual cell and of the body as a whole.

Of further practical importance is the fact that environmental forces can be so focused on the developing embryo as to produce what appears to be a mutation, the so-called phenocopy of Goldschmidt.[15] Recently, Wesselhoeft[16] pointed out that German measles, a relatively mild infection, produces an appalling amount of deficiency in the children of mothers who acquired the infection early in pregnancy. This brings to medical attention the extremely important question of the role of "blastophthoria," a term that means merely sick or injured germ plasm as the source of deficiency in human beings. This concept was introduced by Forel[17] and emphasized by Myerson[18] as a theoretic constitutional basis for mental disease, but the geneticists have rather loftily dismissed it as unimportant. Muller's work, which is the great climax of similarly directed investigations, shows that the germ plasm does not dwell in a sanctum sanctorum, but can be destroyed, injured and deviated from normal functioning by the universe of penetrating forces in which it dwells.[19]

TO THE FELLOWS OF THE MASSACHUSETTS MEDICAL SOCIETY

On behalf of the trustees I welcome to membership in the Boston Medical Library all of you who are not already numbered among its fellows.

The Boston Medical Library is one of the leading institutions of its kind in the country. On its shelves are over 200,000 volumes and its collection of periodicals in many languages is outstanding. We wish this great library to be at the service of every physician in Massachusetts, and the share of your increased membership dues that has been allocated to it will make such a goal possible.

We hope that you will show your interest in the Library by visiting it and making use of its facilities.

WALTER G. PHIPPEN
President

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MASSACHUSETTS MEDICAL SOCIETY

TREASURER'S OFFICE

All members should be reminded that the proportion of the refund returned to each district society is based on the number of dues paid by March 1 in that district, and also that the names of members who have not paid their dues by March 1 are automatically removed from the mailing list of the *Journal* until such dues are paid. This year $8000 is being returned to the district societies, rather than $4000 as heretofore.

ELIOT HUBBARD, JR., *Treasurer*

DEATHS

CRUFF — Frederick E. Cruff, M.D., of Norwell, died on January 18. He was in his fifty-sixth year.

Dr. Cruff received his degree from Harvard Medical School in 1920. He was a member of the Association of Military Surgeons.

His widow and a daughter survive.

HUNT — Ernest L. Hunt, M.D., of Worcester, died on January 17. He was in his seventy-first year.

Dr. Hunt received his degree from Harvard Medical School in 1902. He was surgeon-in-chief of Fairlawn Hospital and director of Worcester Health Department laboratories and was formerly associate medical examiner for Worcester County. He served as a medical officer in World War I. He was a member of the New England Surgical Society and the American Urological Association and a fellow of the American College of Surgeons and the American Medical Association.

His widow, three daughters, a son and nine grandchildren survive.

JONES — Raymond C. Jones, M.D., of Fitchburg, died on December 2. He was in his sixty-seventh year.

Dr. Jones received his degree from University of Vermont College of Medicine in 1902.

His widow and a sister survive.

KING — Connie H. King, M.D., of Duxbury, died on January 22. He was in his forty-first year.

Dr. King received his degree from University of Tennessee College of Medicine in 1929. He was assistant medical examiner for Plymouth County and chief anesthetist at Jordan Hospital, Plymouth.

His widow, a son, a daughter, two brothers and a sister survive.

McMILLAN — Archibald McMillan, M.D., of Athol, died on January 16. He was in his seventy-sixth year.

Dr. McMillan received his degree from Bowdoin Medical School in 1904.

PENDOLA — Anthony S. Pendola, M.D., of Holyoke, died on January 10. He was in his fifty-eighth year.

Dr. Pendola received his degree from Middlesex University School of Medicine in 1920. He was a member of the staffs of Providence and Holyoke hospitals and was assistant city physician.

His widow, three brothers and two sisters survive.

POSTGRADUATE LECTURE COURSE

The third Postgraduate Lecture Course, which has been arranged by the Committee on Postgraduate Medical Education, Massachusetts Medical Society, in co-operation with the Massachusetts Department of Public Health, will begin on March 8. The meetings will be held at Sanders Theater in Memorial Hall, Harvard University, Cambridge. These lectures are designed for all physicians of Massachusetts and surrounding states, medical officers, hospital residents, interns, medical students and postgraduate students.

All those who plan to attend but have not enrolled should do so immediately by either returning the post card recently forwarded to all physicians in Massachusetts or addressing a post card or letter directly to Postgraduate Lecture Course Committee, Massachusetts Medical Society, 8 Fenway, Boston 15.

The detailed program for the course is as follows (no buffet suppers will be served this year):

Monday, March 8. EARLY DIAGNOSIS AND TREATMENT OF CANCER. Chairmen: Charles C. Lund and Ira T. Nathanson.

5:55–6:00. OPENING REMARKS. Edward P. Bagg, president, Massachusetts Medical Society.

6:00–6:40. **Cancer of the Oral Cavity.** William S. MacComb, attending surgeon, Memorial Hospital, New York City.

6:40–7:05. **Cancer of the Larynx and Pharynx.** Leroy A. Schall, chief, Oto-Laryngological Service, Massachusetts Eye and Ear Infirmary; professor of laryngology and otology, Harvard Medical School.

7:05–7:35. **Cancer of the Lung.** E. D. Churchill, chief, West Surgical Service, Massachusetts General Hospital.

7:35–8:05. **Cancer of the Esophagus.** Richard H. Sweet, visiting surgeon, Massachusetts General Hospital; surgeon, Palmer Memorial Hospital; instructor in surgery, Harvard Medical School.

8:05–8:30. **Cancer of the Stomach.** Arthur W. Allen, chief, East Surgical Service, Massachusetts General Hospital; lecturer in surgery, Harvard Medical School.

8:30–9:00. **Cancer of the Colon and Rectum.** Richard B. Cattell, surgeon, Lahey Clinic, New England Baptist Hospital and New England Deaconess Hospital.

Wednesday, March 10. EARLY DIAGNOSIS AND TREATMENT OF CANCER. Chairmen: Charles C. Lund and Ira T. Nathanson.

3:00–3:45. **Cancer of the Breast — Diagnosis and Surgical Aspects.** C. D. Haagansen, assistant professor of surgery, College of Physicians and Surgeons, Columbia University, New York City.

3:35–4:30. **Cancer of the Breast — Radiation Aspects.** M. Lenz, chief, Radiological Department, Presbyterian Hospital, New York City; professor of clinical radiology, College of Physicians and Surgeons, Columbia University, New York City.

4:30–5:15. **Cancer of the Cervix — Early Diagnosis.** Paul Younge, visiting surgeon, Free Hospital for Women, Brookline.

5:15–6:00. **Cancer of the Cervix and Endometrium.** Joe V. Meigs, chief, Gynecological Service, Massachusetts General Hospital; clinical professor of gynecology, Harvard Medical School.

Monday, March 15. CARDIOVASCULAR DISEASES. Chairmen: Paul D. White and Louis Wolff.

6:00–6:25. **Examination of the Patient with Heart Disease.** Louis Wolff, associate in medicine, Harvard Medical School; visiting physician, Beth Israel Hospital; consultant in cardiology, Beth Israel Hospital.

6:25–6:50. **Roentgenology — Its Value in Cardiac Diagnosis.** Felix F. Fleischner, director, X-ray Department, Beth Israel Hospital; instructor in radiology, Harvard Medical School and Tufts College Medical School.

6:50–7:15. **Electrocardiography Today.** Conger Williams, assistant in medicine, Harvard Medical School; consultant in diabetes, Boston Lying-in Hospital.

7:15–7:30. Intermission.

7:30–8:00. **Symptoms of Heart Disease.** S. A. Levine, assistant professor of medicine, Harvard Medical School; physician, Peter Bent Brigham Hospital.

8:00–8:30. **The Use of Digitalis.** A. S. Freedberg, associate visiting physician, associate in medical research, Beth Israel Hospital; associate in medicine, Harvard Medical School.

8:30–9:00. **Treatment of Myocardial Failure.** Paul D. White, clinical professor of medicine, Harvard Medical School; physician, Massachusetts General Hospital.

Wednesday, March 17. EPIDEMIC DIARRHEA IN INFANTS. Chairmen: Clement A. Smith and Nathan B. Talbot.

3:00–3:30. **Present Concepts of Etiology.** John F. Enders, associate professor of bacteriology and immunology, Harvard Medical School; chief, Research Division of Infectious Diseases, Children's Hospital.

3:30–4:00. **Epidemiologic Management.** Clement A. Smith, assistant professor of pediatrics, Harvard Medical School; chief, Infants' Hospital.

4:00–4:30. **Clinical Management.** Allan M. Butler, professor of pediatrics, Harvard Medical School; chief, Children's Medical Service, Massachusetts General Hospital.

4:30–6:00. **Open Forum.** Drs. Butler, Smith, Talbot and Enders.

Monday, March 22. ARTHRITIS. Chairmen: Theodore B. Bayles and Charles L. Short.

6:00–6:20. **Classification of Joint Disease.** Charles L. Short, associate physician, Massachusetts General Hospital.

6:20–6:50. **The Diagnostic Value of Synovial Fluid Examination.** Marian W. Ropes, assistant professor of medicine, Harvard Medical School; associate physician, Massachusetts General Hospital.

6:50–7:20. **Rheumatoid Spondylitis: Recognition and Treatment.** J. Sydney Stillman, assistant in medicine, Harvard Medical School; physician-in-chief, Robert Breck Brigham Hospital.

7:20–7:30. Intermission.

7:30–8:00. **Gout.** Walter Bauer, associate professor of medicine, Harvard Medical School; physician, Massachusetts General Hospital.

8:00–8:30. **Shoulder-Hand Syndromes.** Theodore B. Bayles, assistant in medicine, Harvard Medical School; visiting physician, Robert Breck Brigham Hospital; chief, Arthritis Clinic, Peter Bent Brigham Hospital.

8:30–9:00. **Nonsurgical Orthopedic Care of Arthritic Joints.** Theodore A. Potter, instructor in orthopedic surgery, Boston University School of Medicine; visiting orthopedic surgeon, Robert Breck Brigham and Massachusetts Memorial Hospitals.

Wednesday, March 24. INFECTIOUS DISEASES. Chairmen: Maxwell Finland and Charles A. Janeway.

PART I

3:00–3:20. **Indications and Uses of Vaccines and Antiserums.** Geoffrey Edsall, director, Division of Biologic Laboratories; assistant professor of public-health bacteriology, Harvard School of Public Health.

3:20–3:40. **Hemolytic-Streptococcus Infections.** Louis Weinstein, assistant professor of medicine, Boston University School of Medicine; instructor in infectious diseases, Harvard Medical School; physician and chief-of-service, Haynes Memorial Hospital.

3:40–4:00. **Rheumatic Fever.** David Rutstein, visiting physician, Children's Hospital and House of the Good Samaritan; consultant in preventive medicine, Peter Bent Brigham Hospital and Massachusetts General Hospital; professor of preventive medicine, Harvard Medical School.

4:00–4:15. Discussion.

PART II

4:15–4:35. **Clinical Aspects of Virus Diseases.** Conrad Wesselhoeft, professor of clinical medicine, Boston University School of Medicine; professor of communicable diseases, Harvard Medical School and Harvard School of Public Health.

4:35–4:55. **Newer Methods in the Diagnosis of Specific Virus Diseases.** F. Sargent Cheever, assistant professor of bacteriology, Harvard Medical School.

4:55–5:00. Discussion.

PART III

5:00–5:30. **The Diagnosis of Obscure Fever.** Chester S. Keefer, professor of medicine, Boston University School of Medicine; director, Evans Memorial, Massachusetts Memorial Hospitals.

5:30–6:00. **Progress in Pulmonary Tuberculosis.** Dr. Donald S. King, lecturer in medicine, Harvard Medical School; physician, Massachusetts General Hospital.

Monday, March 29. NEUROLOGY. Chairmen: Derek E. Denny-Brown and Charles S. Kubik.

6:00–6:40. **The Place of Thymectomy in the Treatment of Myasthenia Gravis.** Henry R. Viets, lecturer on neurology, Harvard Medical School; neurologist, Massachusetts General Hospital.

6:50–7:30. **Clinical Varieties of Multiple Neuritis.** Derek E. Denny-Brown. James Jackson Putnam Professor of Neurology, Harvard Medical School; director, Neurological Unit, Boston City Hospital.

7:40–8:20. **The Place of Penicillin in the Treatment of Neurosyphilis.** Raymond D. Adams, assistant professor of neurology, Harvard Medical School; lecturer in neurology, Tufts College Medical School; visiting neurologist and neuropathologist, Boston City Hospital.

8:20–9:00. **Diagnosis of Spinal-Cord Compression.** Charles S. Kubik, chief, Neurological Service, Massachusetts General Hospital; professor of medicine, Harvard Medical School.

Wednesday, March 31. DERMATOLOGY. Chairmen: John G. Downing and Jacob H. Swartz.

3:00–3:30. **Treatment of Bacterial, Virus and Parasitic Diseases of the Skin.** Francis Thurmon, clinical professor of dermatology, Tufts College Medical School.

3:30–4:00. **Treatment of Psoriasis, Seborrhea and Nutritional Diseases.** Bernard Appel, clinical professor of dermatology, Tufts College Medical School.

4:00–4:30. **Treatment of Fungus Diseases of the Skin.** Jacob H. Swartz, assistant professor of dermatology, Harvard Medical School and Postgraduate School.

4:30–5:00. **Dermatologic Allergy, Including Drug Eruptions.** G. Marshall Crawford, acting head, Department of Dermatology, Harvard Medical School.

5:00–5:30. **Cutaneous Manifestations of Systemic Diseases.** John G. Downing, professor of dermatology, Boston University School of Medicine and Tufts College Medical School.

5:30–6:00. Question Period.

Monday, April 5. ALLERGY. Chairmen: Jeremiah E. Greene and Francis C. Lowell.

6:00–6:45. **Allergy — Its Scope and Place in Medicine.** Walter S. Burrage, assistant physician, Massachusetts General Hospital; physician, New England Deaconess Hospital; assistant in medicine, Harvard Medical School.

6:45–7:30. **The Treatment of Asthma with Drugs.** Francis C. Lowell, associate professor of medicine, Boston University School of Medicine; chief, Allergy Clinic, Out-Patient Department, Massachusetts Memorial Hospitals.

7:30–7:40. Intermission.

7:40–8:15. **Skin Tests and Injection Therapy in Hay Fever and Asthma.** Jeremiah E. Greene, affiliated with Massachusetts General Hospital, Mt. Auburn Hospital Newton-Wellesley Hospital and New England Baptist Hospital.

8:15–9:00. **The Antihistaminic Drugs.** Francis C. Lowell, associate professor of medicine, Boston University School of Medicine; chief of Allergy Clinic, Out-Patient Department, Massachusetts Memorial Hospitals.

Wednesday, April 7. OFFICE PROCEDURES IN DISEASES OF MOUTH, VAGINA AND RECTUM. Chairmen: Walter B. Hoover, Roy E. Mabrey and Francis P. McCarthy.

3:00–3:20. **Lesions of the Oral Mucosa.** Francis P. McCarthy, professor of oral medicine, Tufts College Dental School; lecturer in dermatology and syphilology, Tufts College Medical School.

3:20–3:40. **Lesions of the Hard Parts of the Oral Cavity.** Joseph F. Volker, dean and professor of clinical dentistry, Tufts College Dental School.

3:40–4:00. **Lesions of the Oropharynx.** Walter B. Hoover, chief, Department of Nose and Throat, Lahey Clinic.

4:00–4:10. **Intermission.**

4:10–4:35. **The Vaginal Smear in the Diagnosis of Carcinoma of the Uterus.** Howard Ulfelder, assistant in surgery, Massachusetts General Hospital.

4:35–5:00. **Displacements of the Uterus.** Louis E. Phaneuf, professor of gynecology, Tufts College Medical School; chief, Department of Gynecology, Carney Hospital.

5:00–5:20. **Methods of Diagnosis of Anorectal Diseases.** Roy E. Mabrey, assistant surgeon, Massachusetts General Hospital.

5:20–5:40. **The Problem of Rectal Bleeding.** Neil W. Swinton, surgeon, Lahey Clinic, New England Deaconess Hospital and New England Baptist Hospital.

5:40–6:00. **Premalignant and Malignant Lesions of Rectum and Colon.** E. Parker Hayden, chief, Proctologic Clinic and assistant visiting surgeon, Massachusetts General Hospital.

Monday, April 12. CLINICAL, ROENTGENOLOGICAL, PATHOLOGICAL SESSION. Chairman: Merrill C. Sosman (who will present the Case Reports).

6:00–7:00. **Bone Diseases.** William T. Green, orthopedic surgeon, Peter Bent Brigham Hospital; visiting orthopedic surgeon, Children's Hospital; assistant professor of orthopedic surgery, Harvard Medical School; Sidney Farber, pathologist, Children's Hospital; assistant professor of pathology, Harvard Medical School.

7:00–8:00. **Chest Diseases.** Cutting B. Favour, associate in medicine, Harvard Medical School; associate in medicine, Peter Bent Brigham Hospital; and Alan Moritz, pathologist, Peter Bent Brigham Hospital; professor of legal medicine, Harvard Medical School.

8:00–9:00. **Endocrine Problems.** Lewis M. Hurxthal, director, Department of Medicine, Lahey Clinic; and S. Burt Wolbach, pathologist-in-chief, Children's Hospital; Shattuck Professor of Pathology, Harvard Medical School.

Wednesday, April 14. GASTROENTEROLOGY. Chairmen: Benjamin M. Banks and James A. Halsted.

3:00–3:20. **Functional Gastrointestinal Disorders — Principles of Management.** James A. Halsted, physician-in-chief, Faulkner Hospital.

3:20–3:45. **The Role of the Internist in the Management of Chronic Ulcerative Colitis.** Benjamin M. Banks, chief, Gastrointestinal Clinic, and associate visiting physician, Beth Israel Hospital.

3:45–4:10. **The Role of the Psychiatrist in the Management of Chronic Ulcerative Colitis.** Erich Lindemann, psychiatrist, Massachusetts General Hospital.

4:10–4:35. **The Role of the Surgeon in the Management of Chronic Ulcerative Colitis.** Leland S. McKittrick, visiting surgeon, Massachusetts General Hospital.

4:35–4:45. **Intermission.**

4:45–5:10. **The Differential Diagnosis of Dysphagia.** Seymour Gray, senior associate in medicine, Peter Bent Brigham Hospital.

5:10–5:35. **Surgical Treatment of Carcinoma of the Esophagus and Cardiac End of the Stomach.** Richard H. Sweet, visiting surgeon, Massachusetts General Hospital.

5:35–6:00. **Discussion.**

Tuesday, April 20 (Note change from Monday, April 19). ENDOCRINES AND DIABETES. Chairmen: Frank N. Allan and Joseph C. Aub.

6:00–6:30. **Treatment of Diabetic Coma.** Allan M. Butler, professor of pediatrics, Harvard Medical School.

6:30–7:00. **Endocrine Complications of Diabetes.** Frank N. Allan, associate director, Medical Department, Lahey Clinic.

7:00–7:30. **Sex Hormones in Growth.** Joseph C. Aub, professor of research in medicine, Harvard Medical School.

7:30–8:00. **Menstrual Disorders.** John Rock, clinical professor of gynecology, Harvard Medical School.

8:00–8:30. **The Theoretical Background of Therapy of the Thyroid.** J. H. Means, Jackson Professor of Clinical Medicine, Harvard Medical School.

8:30–9:00. **Studies on Acromegaly.** Fuller Albright, associate professor of medicine, Harvard Medical School.

Wednesday, April 21. PSYCHIATRY. Chairmen: Robert E. Fleming and Harry C. Solomon.

3:00–3:30. **Psychiatric Procedure and Technics.** Harry C. Solomon, professor of psychiatry, Harvard Medical School.

3:30–4:00. **Adjustment Problems of the Pre-school Child.** Marian C. Putnam, director, The James Jackson Putnam Children's Center.

4:00–4:30. **Psychiatric Problems of the Adolescent.** George E. Gardner, executive director, Judge Baker Guidance Center.

4:30–5:00. **Psychiatric Problems of Young Men.** Gaylord P. Coon, psychiatrist, Department of Hygiene, Harvard University.

5:00–6:00. **Psychiatric Aspects of Acute Grief.** Erich Lindemann, psychiatrist, Massachusetts General Hospital.

Monday, April 26. CONVULSIVE SEIZURES. Chairmen: William G. Lennox and Maxwell E. Macdonald.

6:00–6:30. **Differential Diagnosis and Etiology.** William G. Lennox, visiting neurologist, Boston City Hospital; assistant professor of neurology, Harvard Medical School.

6:30–7:10. **Neurosurgical Considerations and Treatment.** Wilder Penfield, Montreal, Canada, professor of neurology and neurosurgery, McGill University, Montreal.

7:10–7:50. **Medical and Drug Therapy.** H. Houston Merritt, professor of clinical neurology, Columbia University College of Physicians and Surgeons, New York City.

7:50–8:20. **Psychological and Social Problems and Treatment.** Maxwell E. Macdonald, professor of neurology, Harvard Medical School; senior neurologist, Boston City Hospital.

8:20–9:00. **Question Period.**

Wednesday, April 28. Blood Diseases. Chairmen: William Dameshek and John W. Norcross.

Part I. The Diagnosis and Treatment of Hemorrhagic Disease.

3:00–3:30. **Diagnostic Tests.** Jacob Neber, assistant in hematology, Joseph H. Pratt Diagnostic Hospital.

3:30–4:00. **The Purpuras.** Charles S. Davidson, associate in medicine, Harvard Medical School; junior visiting physician, Boston City Hospital.

4:00–4:30. **Hemophilia.** Benjamin Alexander, instructor in medicine, Harvard Medical School; visiting physician and associate in medical research, Beth Israel Hospital.

Part II. Symposium on Treatment.

4:30–5:00. **Treatment of Anemia.** Donat Cyr, assistant hematologist, Department of Medicine, Lahey Clinic.

5:00–5:30. **Chemotherapy of Leukemia and Lymphoma.** William Dameshek, professor of clinical medicine, Tufts College Medical School; consultant in hematology and visiting physician, Joseph H. Pratt Diagnostic Hospital.

5:30–6:00. **Splenectomy in Blood Disorders.** John W. Norcross, hematologist, Department of Medicine, Lahey Clinic.

Monday, May 3. Genitourinary Disease. Chairmen: J. Hartwell Harrison and George C. Prather.

6:00–6:30. **Hematuria: Significance and Management.** J. Hartwell Harrison, senior associate in genitourinary surgery, Peter Bent Brigham Hospital; associate professor of genitourinary surgery, Harvard Medical School.

6:35–7:00. **Pain: Interpretation relative to genitourinary causes.** George C. Prather, visiting neurologic surgeon, Boston City Hospital; instructor in genitourinary surgery, Harvard Medical School.

7:10–7:40. **Streptomycin in the Treatment of Nontuberculous Bacillary Urinary Infections.** Maxwell Finland, physician-in-chief, Fourth Medical Service, Boston City Hospital; assistant professor in medicine, Harvard Medical School.

7:45–8:15. **The Diagnosis and Treatment of Cancer of the Prostate.** Wyland F. Leadbetter, assistant professor in genitourinary surgery, Harvard Medical School; clinical professor of urology, Tufts College Medical School; associate visiting urologist, Massachusetts General Hospital.

8:20–8:50. **Present Outlook in Carcinoma of the Bladder.** Roger C. Graves, clinical professor of urology, Tufts College Medical School; urologist-in-chief, Carney Hospital.

Wednesday, May 5. Obstetrics. Chairmen: Duncan E. Reid and Benjamin Tenney, Jr.

Part I. Diabetes Mellitus in Pregnancy.

3:00–3:25. **Medical Treatment.** David Hurwitz, assistant instructor in medicine, Harvard Medical School; consultant in diabetes, Boston Lying-in Hospital.

3:25–3:45. **Endocrinological Treatment.** Olive Smith, Ph.D., director, Fearing Research Laboratory, Free Hospital for Women.

3:45–4:00. **Question Period.**

Part II. Heart Disease in Pregnancy

A twenty-five-year review

4:00–4:25. **Treatment.** Burton Hamilton, clinical associate, Thorndike Memorial Laboratory, Boston City Hospital; instructor, graduate courses, Harvard Medical School.

4:25–4:30. **Question Period.**

Part III. Hypertension in Pregnancy.

4:30–4:55. **Medical Aspects.** Lewis Dexter, associate in medicine, Peter Bent Brigham Hospital and Harvard Medical School.

4:55–5:15. **Obstetrical Aspects.** Benjamin Tenney, associate professor of obstetrics, Boston University School of Medicine; assistant obstetrician, Massachusetts General Hospital.

5:15–5:30. **Question Period.**

Part IV. Vaginal Bleeding in Last Trimester of Pregnancy.

5:30–5:45. **Clinical Aspects.** H. Bristol Nelson, senior obstetrician, Boston Lying-in Hospital; professor of gynecology and obstetrics, Harvard Medical School.

5:45–5:55. **Pathological Aspects.** Arthur T. Hertig, assistant professor of obstetrics and assistant professor of pathology, Harvard Medical School; pathologist, Free Hospital for Women; obstetrician, Out-Patient Department, Boston Lying-in Hospital.

5:55–6:00. **Question Period.**

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

REVIEW OF COMMUNICABLE DISEASES IN 1947

The year 1947 was very favorable regarding the general prevalence of communicable diseases. The total number of cases reported to the Department—76,469—was the lowest reported since 1916, the lowest figure in recent years being 78,089 cases in 1938. The low figure for 1947, however, can be attributed largely to the low prevalence of some of the less serious diseases of childhood. Both measles and German measles were much below normal. Scarlet fever reached the lowest level since 1905. On the other hand, the record for 1947 would have been still lower had not chicken pox reached the second highest figure since the disease was reportable, and mumps and whooping cough maintained about the normal prevalence.

Meningococcal meningitis, lobar pneumonia and typhoid fever were at very low levels, and gonorrhea and syphilis showed some decline. Bacillary dysentery, poliomyelitis and salmonellosis were at about the normal prevalence. On the other hand, diphtheria, tularemia, trichinosis and undulant fever all showed an increase.

Diphtheria continues to be a trying problem. It was hoped that the increased emphasis on immunization programs would bring the prevalence down, but more cases were reported this year than in 1946, which itself was the highest year since 1934.

On the other hand, not a single case of smallpox was reported, marking the fifteenth year since the disease was prevalent in the State. Typhoid fever dropped to the second lowest level since reporting began.

Disease	1947	1946	Seven-Year Median
Actinomycosis	1	3	3
Anthrax	2	2	5
Chancroid	31	19	20*
Chicken pox	18,793	11,912	11,912
Diphtheria	442	439	154
Dog bite	11,148	11,559	10,701
Dysentery, amebic	10	7	4
Dysentery, bacillary	188	68	245
Encephalitis, infectious	9	8	21
German measles	932	4,618	2,334
Gonorrhea	3,805	5,062	4,701
Granuloma inguinale	5	4	4*
Hookworm	2	3	0
Lymphocytic choriomeningitis	7	4	4†
Lymphogranuloma venereum	8	10	31*
Malaria	91	509	116
Measles	12,069	38,400	22,338
Meningitis, meningococcal	52	116	161
Meningitis, Pfeiffer bacillus	48	38	32
Meningitis, pneumococcal	28	48	48†
Meningitis, other forms	12	10	25†
Meningitis, undetermined	59	44	44†
Mumps	8,383	5,810	10,491
Ophthalmia neonatorum	599‡	485‡	593‡
Pneumonia, lobar	1,344	1,483	3,104
Poliomyelitis	346	378	252
Psittacosis	0	2	0
Rabies in animals	0	1	5
Rocky Mountain spotted fever	3	1	0
Salmonellosis	156	180	120
Scarlet fever	4,428	6,079	10,373
Septic sore throat	112	179	179
Syphilis	3,211	4,970	5,024
Tetanus	11	13	14
Trachoma	7	2	9
Trichinosis	59	38	31
Tuberculosis, pulmonary	2,640	2,858	2,816
Tuberculosis, other forms	190	179	199
Tuberculosis, hilus	8	10	15
Tularemia	7	2	1
Typhoid fever	25	33	34
Typhus fever	0	3	2
Undulant fever	76	52	49
Whooping cough	7,125	6,514	7,445

*Three-year median.

†Five-year median.

‡Includes suppurative conjunctivitis.

MISCELLANY

APPOINTMENT OF MANFRED BOWDITCH

Manfred Bowditch, formerly field director of the Saranac Laboratory and director of the Division of Occupational Hygiene of the Department of Labor and Industries of the Commonwealth of Massachusetts, has been appointed director of health and safety of the Lead Industries Association. He will make his office at the Association's headquarters, 420 Lexington Avenue, New York City.

NATIONAL COMMITTEE FOR MENTAL HYGIENE

Dr. Arthur H. Ruggles, former superintendent of the Butler State Hospital, Providence, Rhode Island, has been elected president of the National Committee for Mental Hygiene. Dr. Ruggles, who has been the superintendent of Butler Hospital since 1922, is president of the Emma Pendleton Bradley Home, Providence, and a trustee of Dartmouth College. He is consultant in mental hygiene in the Department of University Health and lecturer in psychiatry at Yale University, a member of the advisory council on research in nervous and mental diseases and consultant, United States Public Health Service, and a member of the advisory council of the Department of Social Welfare of Rhode Island. Dr. Ruggles is a former president of the American Psychiatric Association and of the New England Society for Psychiatry.

CANCER

A new journal, *Cancer*, sponsored by the American Cancer Society, will make its appearance this spring. Every phase of the cancer problem will be covered, with major emphasis on clinical aspects. Dr. Fred W. Stewart, of Memorial Hospital, New York City, will be the editor-in-chief, assisted by an editorial advisory board. Original papers should be submitted for consideration to the editor, Dr. Fred W. Stewart, 444 East 68th Street, New York 21, New York. *Cancer* will be published bimonthly, at eight dollars a year, by Paul B. Hoeber, Incorporated, Medical Book Department of Harper and Brothers.

CORRESPONDENCE

EFFECT OF ENVIRONMENTAL INFLUENCES

To the Editor: Just about twenty-five years ago Professor Hermann J. Muller read a paper at the Second International Congress of Eugenics demonstrating that external influences do not increase the mutation rate in animals — in other words, that they have little to do with hereditary processes.

Last year the same distinguished scientist received the Nobel prize for proving that environmental influences, especially radiations of various types, enormously increase the mutation rate. This in itself is of human interest in that a man completely changed his opinion in twenty-five years, but what is more important is that Professor Muller has a great deal to say about the danger to the human race from radiations and radioactive substances of all kinds. Since most of the mutations are either lethal or defective, it seems that the damage need not appear at once. His article in the September number of the *Journal of Heredity* culminates in a very portentous warning to the medical profession that the use of radioactive substances may be associated with harm to the race of a kind not at all apparent at once. In fact, the front cover of the journal is wholly given over to this warning.

Every man who undertakes the use of radioactive substances of any kind, whether for therapy or investigation, should read this great geneticist's paper, which, incidentally, was read when he received the Nobel prize. He speaks not only of the dangers of x-rays but also of mustard gas and of ultraviolet radiation. We are going ahead rather fast in the use of radiated atoms and molecules in the treatment of disease and also in the investigation of physical processes. It might be well to study the germ plasm — that is, the substance of ovary and testicle — at the same time that studies are being carried out on the thyroid gland or whatever part of the body is being bombarded for therapeutic or investigative purposes.

Medicine is in a dangerous position. It has at hand powerful instruments, which it often uses without due regard for the welfare of the patient as a whole. Similarly, the use of mineral oil for constipation has been assumed to be an innocuous way of regulating one of the functions of the gastrointestinal tract. Now it appears likely that this is a way of preventing the absorption of vitamin A.

Abraham Myerson, M.D.

171 Bay State Road
Boston

DEPRIVATION OF LICENSE

To the Editor: At the meeting of the Board of Registration in Medicine held January 15, the registration of Dr. Daniel Kaplan of Worcester was suspended for three months because of gross misconduct in the practice of his profession.

H. Quimby Gallupe, M.D., *Secretary*
Board of Registration in Medicine

State House
Boston

BOOK REVIEW

Human Genetics. By Reginald R. Gates, B.Sc., Ph.D., D.Sc., LL.D. 8°, cloth, two volumes, 1518 pp., with 325 illustrations. New York: Macmillan Company, 1946. $15.00.

This monumental treatise is essentially a reference work. It is based on the author's previous book, *Heredity and Eugenics*, published in 1923, but the number of chapters has been doubled and the amount of material increased fivefold. The relation of heredity to all diseases and abnormalities is discussed in detail. The first chapters deal with the general principles of heredity in man, human cytology and linkage. These are followed by special chapters on the diseases and abnormalities of the various organs and systems, and on general diseases and conditions. Of special interest are the chapters on color blindness, albinism, metabolic defects,

hemophilia, blood groups, allergy, hereditary syndromes, sexual and intersexual conditions, twins and twinning, cancer, constitution, body build and susceptibility, stature and size and anthropologic characters. The text is well arranged and well written in an easy style. Selected references are appended to each chapter. A comprehensive index concludes the work. The book is well published in every way. It is a delight to read the handsome, clear type on good paper. The treatise is recommended for all medical, scientific, college and public libraries and should prove useful to all physicians.

NOTICES

ANNOUNCEMENTS

Dr. Louis S. Chase announces the removal of his office to 416 Marlborough Street, Boston.

Dr. Clarke Staples announces the removal of his office to 543 Highland Avenue, Malden.

Dr. Donald M. Stewart announces the opening of an office for the practice of obstetrics and gynecology at 745 Main Street, Fitchburg.

NEW ENGLAND HEART ASSOCIATION

The next meeting of the New England Heart Association will be held in the amphitheater of the Dowling Building, Boston City Hospital, on Monday, March 1, at 8:15 p.m. Dr. Laurence B. Ellis will preside.

PROGRAM

Experience with Dicumarol in Acute Myocardial Infarction. Drs. E. Bresnick, L. B. Ellis, H. N. Hultgren, B. Rapoport and H. S. Sise.

Venous-Pressure Responses to Exercise and Abdominal Compression. Dr. H. N. Hultgren.

Observations on the Heart in Women. Dr. B. E. Hamilton.

Circulatory Dynamics in Myxedema. Drs. R. A. Bloomfield, L. B. Ellis, W. K. Long, G. Maresh, J. G. Mebane and J. P. Milnor.

Evaluation of Vitamin E in the Treatment of Angina Pectoris. Drs. I. B. Ravin and K. H. Katz.

Two to Three Year Follow-up on Penicillin-Treated Cases of Subacute Bacterial Endocarditis. Dr. B. Rapoport.

Interested physicians and medical students are cordially invited to attend.

APPOINTMENT OF COMMISSIONED OFFICERS IN NAVY MEDICAL AND DENTAL CORPS

The statutory authority contained in Public Law 365 of 80th Congress, Title II (Army–Navy–Public Health Service Medical Officer Procurement Act of 1947), makes it possible for civilian doctors to become commissioned officers in the regular Navy, with ranks up to and including that of captain, provided they meet the professional and physical qualifications.

To qualify for appointment a doctor must be a citizen of the United States and a graduate of a Class "A" medical school and must have served at least one year's internship in an approved hospital. Candidates will then be judged on a number of qualifications, such as membership in a specialty board, teaching connections, the number of years of professional or scientific practice and hospital or laboratory connections.

Interested physicians should apply to the Bureau of Naval Personnel, Bureau of Medicine and Surgery, Navy Department, Washington, D. C.

MEDICAL AND SURGICAL POSITIONS IN OVERSEAS ARMY HOSPITALS

Opportunities for advanced training and experience in the various special fields of medicine and surgery in overseas Army hospitals are available. These hospitals are registered with the American Medical Association, and the training may be acceptable by the specialty board as part of the period usually required to be spent in limited practice and experience prior to admission for examination. Interested members of the medical profession who have completed the formal training requirements for certification in one of the special fields are eligible to apply for these positions.

For information concerning the specialties and the location of hospitals, as well as the terms under which physicians may avail themselves of the training, application should be made to the Surgeon General, United States Army, Washington 25, D. C.

NATIONAL TUBERCULOSIS ASSOCIATION TEACHING AND RESEARCH FELLOWSHIPS

National Tuberculosis Association teaching and research fellowships in the field of tuberculosis are available, it was recently announced. Annual stipends for the fellowships will range from $2400 to $3200. Provision will also be made for laboratory fees and similar incidental expenses. The fellowships will be limited to graduates of American schools for teaching and investigation in the United States. Although preference will be given to applicants with a degree of Doctor of Philosophy or Doctor of Medicine, fellowships will not be restricted to the holders of these degrees. Applications will be considered in the fields of pathology and bacteriology, clinical medicine, epidemiology and social and statistical research. Applicants may elect the institutions in which they wish to study.

Persons interested in obtaining a fellowship should write to Dr. James E. Perkins, managing director, National Tuberculosis Association, 1790 Broadway, New York 19, New York, for further information.

KENFIELD MEMORIAL FUND SCHOLARSHIP

A scholarship of $100 will be available in 1948 for training of teachers of deaf adults. Applicants, in addition to being adult residents of the United States should meet the following requirements: personal characteristics necessary for successful teaching; ability to read lips as certified upon examination by an approved instructor in lip reading; a bachelor's degree, or two years of college work or successful experience in teaching in public or private schools, in addition to twelve semester hours of work in adult education, psychology of the handicapped, voice production and control, social service or kindred subjects; and thirty clock hours of private instruction under an approved teacher of lip reading or sixty clock hours of instruction in public-school classes under an approved teacher of lip reading. The winner of the scholarship may take the normal course from any normal training teacher or school or university in the United States offering a course acceptable to the Teachers' Committee of the American Hearing Society. The applicant for the scholarship must be a prospective teacher. Applications from those who are teaching lip reading now cannot be considered. The scholarship must be used within one year from the granting of the award.

Applications must be filed between March 1 and May 1, 1948, with Miss Rose V. Feilbach, 2431 14th Street, N. W., Washington 9, D. C.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, FEBRUARY 26

FRIDAY, FEBRUARY 27

*9:00–10:00 a.m. Clinicopathological Conference. Drs. Chester S. Keefer and H. E. MacMahon. Joseph H. Pratt Diagnostic Hospital.

*10:00 a.m.–12:00 m. Medical Staff Rounds. Peter Bent Brigham Hospital.

MONDAY, MARCH 1

*12:15–1:15 p.m. Clinicopathological Conference. Peter Bent Brigham Hospital.

TUESDAY, MARCH 2

12:00 m. X-Ray Conference. Margaret Jewett Hall, Mt. Auburn Hospital, Cambridge.

*12:15–1:15 p.m. Clinicoroentgenological Conference. Peter Bent Brigham Hospital.

*1:30–2:30 p.m. Pediatric Rounds. Burnham Memorial Hospital for Children, Massachusetts General Hospital.

(*Notices concluded on page xvii*)